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IMMUNIZATION AGAINST DIPHTHERIA AND SCARLET FEVER.[1]

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MAY I first, on behalf of the Health Committee of the Melbourne City Council, and of their Medical Officer of Health, Dr. Dale, express their sincere appreciation of the honour you have done them in asking me to come over and read this paper?

INTRODUCTION.

In entering upon a discussion of the methods of controlling the two chief infectious diseases, diphtheria and scarlet fever, it is, I think, no longer necessary to defend the theory or practice of immunization. Both theoretically and practically it is so well based and firmly established that we are now concerned rather with organization and refinements of technique than with demonstrating principles that have long been accepted. For all these methods depend on the fundamental principle that living tissue reacts against a breach of its integrity by producing specific antibodies against the foreign invader.

As Bordet said, "life is the maintenance of an equilibrium which is constantly threatened". From the age-old struggle between the higher forms of life and the lower has emerged, not one side triumphant and victorious and the other laid low for ever, but a compromise, a *modus vivendi*, whereby the parasite and the host live side by side. But this is not as amicable an arrangement as it sounds—there is a state of constant vigilance on the part of the higher forms and an amazing energy in taking a mean advantage on the part of the lower

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on April 24, 1935.

forms of life, and so the defensive powers of man and animal are always on the alert. When they are so adjusted that they at least balance the invasive powers and virulence of the attacking organism, we have achieved a state of active immunity.

This state of active immunity is to be distinguished from that of passive immunity, such as can be conferred for a brief period by the injection of antibodies—for instance, antitoxin—elaborated in the body of another animal. Passive immunity against diphtheria usually exists in infants and is derived from antibodies in the placental blood, and later in the maternal milk. The difference between this passive immunity and that elaborated in the body of the reacting animal in response to attack is that passive immunity is transient. Active immunity is a property of the reacting tissues and is practically permanent.

Disease is an extreme form of the infective process, when the symbiosis between host and parasite leads to injury of the tissues of the host. Perhaps, by the way, we should also extend our sympathy to the suffering parasite, as Bernard Shaw suggests in his play "Too True to be Good", by his absurd picture of the martyred measles "Microbe". That the symbiosis is wider than we ever dreamt of is shown by our studies of diphtheria and scarlet fever, where many forms of latent or cryptic association occur.

Individual active immunity implies absolute or relative freedom from disease under exposure to risk.

The survival after an attack of disease is by no means the only road to acquired resistance. Probably the most important factor is the occurrence of infections so slight and transient as to escape notice—the "sub-infective" dose of every possible kind of germ that can be interchanged under the usual conditions of human association, particularly among dwellers in cities.

The principle underlying the methods of producing acquired active immunity is to follow these processes of Nature and induce the development of antibodies by stimulating the defensive forces of the body with an "attenuated virus", as Pasteur calls it. When it is left to Nature, many develop immunity, but many fall by the way and succumb to the disease. The production of immunity without the clinical symptoms of disease is the aim of all immunological methods.

DIPHTHERIA.

In the case of diphtheria we are able, by the methods at our disposal, to study the relationship of parasite and host with a degree of accuracy that has not yet been attained with any other infectious disease. Swabbing, virulence tests and Schick tests all give us information from different angles and enable us to follow the spread and mode of action of the parasite.

In our comparatively small population in the City of Melbourne, of less than 100,000 inhabitants, we have had the opportunity of observing these relationships for several years. Where swabbing is necessary because of local prevalences we have also in many cases Schick test records in these children to compare with our results, and, where considered necessary, virulence tests are carried out at the university.

Though the number of individuals treated is only something over 20,000, we have records of all who have been tested and immunized, and can compare these with the notified cases of diphtheria.

In short, our sins are very likely to find us out.

The Schick Test.

It was suggested in 1905, by Theobald Smith, that human beings might be immunized; but this was not practicable until the discovery in 1910, by Bela Schick, of Vienna, that a skin test could be devised which would separate those susceptible to diphtheria from those who were naturally protected.

Titration of the blood of reactors and non-reactors against a measured quantity of toxin by what is called the Romer technique has demonstrated the presence of antitoxin in the blood of non-reactors to the Schick test and none, or very little, in the blood of reactors.

In America a vast amount of evidence has been collected illustrating the connexion between the presence of natural antitoxin of a required concentration in those immune to diphtheria and the absence of antitoxin, as demonstrated by the Schick test and the titration test, in those who contract the disease. We do not know why Nature has fixed this particular limit, but observations on which the Schick test is based have shown that if less than one-thirtieth of a unit of antitoxin per cubic centimetre is present in the circulating blood, the subject is likely to develop clinical diphtheria if exposed to infection.

But besides this quantitative factor—the presence of a minimum quantity of antitoxin amounting to about 200 antitoxin units in the adult—the individual has acquired an added power, that of producing further supplies of antitoxin in response to the stimulus of infection. The Schick test is an indication that the organism has, as one may say, "learnt the trick" of producing this specific antibody to invasion by the diphtheria bacillus, and is able to produce more when attacked.

To test the reliability of the Schick test, nine bacteriologists in New York, four non-reactors and five reactors, submitted to throat swabbing with virulent cultures. All the reactors developed diphtheria and, while one or two of the non-reactors became carriers, none developed the clinical disease.

The technique of the test is quite simple. It consists of the intradermal injection of a small measured quantity of diluted diphtheria toxin. The chief point to be noted is the extreme instability of the diphtheria toxin after it has been diluted, so the solution should always be used immediately. Undiluted, as issued in its capillary tubes, the toxin is remarkably stable and will give accurate results after many months.

The solution now used is eleven cubic centimetres of normal saline solution without antiseptic, which would interfere with its potency. So sensitive is the toxin that it would also be destroyed if the syringe and needle were sterilized with spirit and not properly rinsed, and this is in fact an occasional source of error.

In emptying the tube into the saline solution I prefer to use a rubber bulb or else to discharge it against the side of the bottle by capillary attraction, in either case rinsing the tube thoroughly with the saline solution. If it is crushed, the fragments of fine glass are apt to give trouble with the syringes.

"Record" or "Astra" syringes, and fine, short bevelled needles are most satisfactory. The needle is inserted parallel with the skin of the flexor surface of the forearm, and when the bevel is just buried in the upper layers of the skin, 0·2 cubic centimetre of the solution is introduced. The result is a small white bleb, with a characteristic pitted appearance, caused by the hair follicles and sweat glands, which are still anchored to the deeper layers. If the injection is too deep, this pitted appearance is absent, and it is wise to repeat the dose a little more superficially.

On the other arm the same quantity of a control solution is introduced. This is an exactly similar dilution of the diphtheria toxin heated to 70° C. for five minutes to destroy its toxicity. As a matter of convenience we omit this control test, as we have found that the Moloney test, which will be described later, gives similar information and saves an unnecessary process.

The diluted toxin of the Schick test is, of course, being employed in a rough titration experiment against the antitoxin concentration of the blood. As various values may exist, from zero to one-thirtieth of a unit, which is the minimum required for neutralization, so the test reaction will vary somewhat in intensity.

The response to the Schick test usually arises rather slowly in uncomplicated reactions. It is at its maximum from the fifth to the seventh day, and in definite cases resembles a small superficial burn, perhaps most like sunburn, and as it fades there is usually some desquamation. Roughly, the usual *plus* is recorded for a Schick reaction measuring from 2·0 to 2·5 centimetres and vivid in colour. Above and below we have a *plus plus*, *plus* M. (modified), *plus* F. (faint), indicating various degrees of response. It is well to have these records as exact as possible. A faint reaction may be regarded as practically insignificant, though the individual may have the bad luck later to strike a massive infection and go down to it. I usually immunize these children showing a faint reaction, as, being possessed of a considerable residual immunity, they respond rapidly and effectively. In horses, as you know, those with some original antitoxin are best antitoxin producers.

If a pseudo-reaction is shown, the rate at which it develops is sufficient to distinguish it from a true Schick reaction. In such a case both the test and control arms show inflamed areas, which reach their maximum quickly and fade rapidly after the third day. If the individual is not sensitive to the Schick test, both tests fade at equal rates, and in a week very little is left. But if he is also a Schick reactor, as the control fades the test arm shows an increasing area of redness that is usually in definite contrast with the faded, often bluish, spot on the control arm. Test and control should always be considered together. I do not find that desquamation is confined to the true Schick test. In a very marked pseudo-reaction there may also be some slight peeling on both arms.

Reading of Results of Tests.

Four types of reactions are found when test and control are applied in this way.

1. No reaction is shown on the control arm in twenty-four to forty-eight hours, nor on the test arm in one week. These persons are "naturally" immune to diphtheria and do not require immunization. These are the so-called "negative" reactors.

2. A reaction is shown on the control arm and also on the test arm in twenty-four to forty-eight hours, but at the end of a week this has faded on both arms. These are the pseudo-reactors and do not require immunization. The difference between these and Group 1 is that, in addition to their immunity, they have also developed an allergy to the non-toxic factor in the test material and have thus become sensitized. About 37% of our non-reactors showed this phenomenon.

3. There is no reaction on the control arm, but in five to seven days a red spot of greater or less intensity is shown on the test arm. These individuals do not possess the minimum amount of residual antitoxin necessary to protect them against an attack of diphtheria and require to be artificially immunized.

4. A reaction is shown on both arms in twenty-four to forty-eight hours. In one week the reaction on the control arm has faded; but that on the test arm has increased and is visible as a much brighter spot than the faded one on the control arm. These are not immune to diphtheria, but are probably partly immune, because the sensitivity is usually an indication that they have already met the germ. This group comprises only 4% to 5% of the reactors in our series, and they are either immunized very carefully or left alone, if the sensitivity is marked, so as to avoid probable allergic phenomena, which might be of some severity. They may be immunized by other materials, for example, toxin-antitoxin floccules.

In our series of 5,644 persons recently subjected to primary Schick tests at all ages, 48·5% were non-reactors, and therefore naturally immune, and 52·5% were reactors and required immunization.

Reaction in Relation to Age.

Infants rarely show any reaction to Schick tests under six months of age. This is usually described as being due to an inherited passive immunity from the mother; but another explanation is that there

is some unknown factor which makes skin reactions in infants unreliable. Though it is rare, deaths do occasionally occur from diphtheria in children of this age. Over six months of age the percentage of reactors rises rapidly so that a maximum of susceptibility is established between nine and twelve months. For this reason one does not subject the infant to the unnecessary manipulation involved in testing, but proceeds straight away to immunization. In our analysis of over 5,000 Schick tests the percentage dropped from about 70 in the first five years of age to 37 by the time the tenth year was reached. This was in a population where, of recent years, the germ has been very prevalent and considerable immunity seems to be acquired by the end of school life. It is a clear indication that the most important phase of preventive work is that among infants and children of pre-school age. In children under six years of age it is usually not necessary to use the Schick test before immunization, and I do it only if they are known to have been recently exposed to infection.

In our experience a change of reaction in the naturally immune is very rare, and in 832 retests on 365 primary non-reactors to the Schick test, only five showed a reaction, with three doubtful readings.

Anatoxin.

Although theoretically the ideal antigen for immunization would contain bacterial elements as well as exotoxin, so far no preparation of a diphtheria vaccine has shown to have any antigenic power. Ana-vaccines, a combination of dead bacteria and detoxicated toxin, are freely used in veterinary work, but in producing an antigen against diphtheria, it is only the toxin which has so far proved effective. The reason, of course, why one would like to produce an antibacterial vaccine is to eliminate the carrier state, which usually coexists with a high degree of antitoxic immunity.

In horses, of course, for the production of antitoxin, pure toxin in increasing doses was first employed. In human beings some degree of detoxication of this material was necessary, and this was first produced by mixing toxin with antitoxin in a slightly under-neutralized mixture.

The disadvantages were that if the toxin were under-neutralized, reactions were likely to occur; while, if it were over-neutralized, it was slower and less effective. Also the mixture contained horse serum and there was a danger of sensitization to the serum-protein, which might lead to allergic reaction if any form of serum treatment was required later.

For these reasons, even before the disaster at Bundaberg in 1928, we had been exploring the possibilities of using a different preparation, originated by Ramon, of the Pasteur Institute, and known as anatoxin, which was being used with great success on the Continent and in Canada. Dr. Morgan, of the Commonwealth Serum Laboratories, informed us in 1927 that anatoxin had been in use for some time in the immunization of horses and it was then being prepared for human immunization.

The suspension of all the work in 1928 gave the impetus to the investigation of anatoxin.

In 1923 Ramon had used formalin for detoxicating toxin. When toxin and antitoxin are mixed, flocculation occurs at the point of neutralization. When a given quantity of toxin is mixed with solutions of a standard antitoxin in increasing strengths, the point at which flocculation first occurs is noted. For instance, if four cubic centimetres of toxin first neutralize a solution containing 44 units of antitoxin, the flocculating index is 11. This is also a measure of the antigenic power of the toxin.

Ramon took a toxin of satisfactory toxicity, added to it about 0·3% of formalin, and stored the mixture for three weeks at 37° C. He found that it retained its power of flocculation with antitoxin, but that its toxic power had almost entirely disappeared. A flocculation test, as it is called, corresponding to at least eight to ten antigenic units, is required for successful results. This material, though innocuous to animals, retained its immunizing power. A guinea-pig which would succumb to a minimum lethal dose of toxin survived an equivalent of 500 minimum lethal doses of detoxicated toxin and was afterwards resistant to an almost equal amount of pure toxin.

Ramon called this material anatoxine, but in America it is called formol-toxoid, and probably the latter name is to be preferred. The similarity between the names anatoxin and antitoxin, particularly with the emphasis given it in its English pronunciation, is somewhat confusing, and that this danger is not altogether absent, even in its French form, is illustrated by a rather ghastly story.

In a village in France a man contracted diphtheria and his doctor sent to the nearest chemist for antitoxin. The chemist, with obliging readiness, replied that he had no antitoxin but would anatoxin do instead? His medical advisers decided to try it, and they injected into the abdominal muscles of the patient at least 10 cubic centimetres of a material of which we give only 0·5 cubic centimetre as a first dose. The patient promptly collapsed. However, he was brought round, and the next day, with an intrepidity of which only the French mind could be capable, they gave him another shot. That he survived was probably a tribute to his own constitution as well as to the comparative safety of the material. But I think you will agree that it is not an experiment one would care to repeat.

When we began to use it we were somewhat perturbed by reading in the current literature of reactions, local and general, sometimes of considerable severity in older children, which have been experienced by workers on the Continent. Again, the superior logical qualities of the Gallic mind were displayed, or perhaps they have more faith in their doctors, because in France particularly the general public seemed to take heroic measures, such as we should never dream of applying here, with perfect *sang froid*.

However, in Canada, Dr. Fitzgerald and Dr. Moloney devised a test to exclude those sensitive

or allergic to the material, and therefore likely to react. Our first step was to establish the relationship of this allergy to susceptibility to diphtheria as shown by the Schick test. If it were marked in Schick reactors we should have hesitated to use the material, but the first tests showed that though about one-third of the immunes showed this sensitivity, it was very rare in Schick reactors, and therefore it was justifiable to begin immunization with the new material.

The Moloney Test.

The technique of the Moloney test is to dilute anatoxin with saline solution to a strength of 1 in 20. The saline solution is usually combined with antiseptic, and for large numbers I use 9·5 cubic centimetres of saline solution mixed with 0·5 cubic centimetre of anatoxin. Of this, 0·1 cubic centimetre is injected into the control arm, and the reaction is read in twenty-four to forty-eight hours. The appearance of a red spot, usually slightly raised, distinguishing it from a Schick reaction, which is usually flat and superficial, in size from 1·5 to 2·0 centimetres, is an indication of sensitivity.

The reason why we substituted this test for the control by heated Schick testing material was that, after comparing the reactions in a large number of cases, we found that the same individuals always reacted to both tests and, therefore, although the actual concentration of the Moloney test is about eight times that of the original control, they can be regarded as both giving an index of sensitivity.

It is easy to see that, both being solutions of detoxicated toxin, one being detoxicated by heat, the other by formalin, the reactions should be similar. Also, by doing tests with broth controls, we found that the reaction was not due to the original constituents of the broth. The fact that a reaction to this test is so much more common in those who are immune to diphtheria is regarded as an indication that the sensitivity is really due to some product of the diphtheria bacillus itself. This was confirmed in our series of tests by the interesting observation that hand in hand with the development of immunity with age a lesser, but quite definite, degree of sensitization was observed.

In some hundreds of babies on whom, at first, I did the Moloney test as a safeguard, though naturally omitting the Schick test, I found no reaction at all. As the ages increased, so did the percentage of reactors, and a chart of the Schick and Moloney tests shows them crossing at about twelve years of age.

This means that there is a double response in many people to attack by the diphtheria bacillus: one, the production of antitoxin, and the other, development of sensitivity. This happens also during artificial immunization, and in 500 retests after treatment 30% originally not sensitive to anatoxin had developed an allergic response.

Immunization.

Our methods of immunization depend on the age of the subject. In babies under three, after having established the extreme rarity of sensitivity, we proceed to immunization without a preliminary Moloney test. A small dose is given, from 0·2 to 0·3 cubic centimetre, according to age, as a preliminary, and if no reaction is shown, this is followed by 0·5 and 0·8 cubic centimetre at intervals of about four weeks. This procedure is so simple and so safe that we unhesitatingly recommend its adoption by all family physicians for the infants under their care.

In children between three and six years of age a Moloney test is done and three doses are then given. Ramon first advised two doses, 0·5 and 1·0 cubic centimetre, but in young children where there has as yet been no primary stimulus and where there may be no secondary stimulus for some years, we decided very early to give three doses, although two doses give a very fair protection. We have had four cases of diphtheria after two doses in toddlers, but so far none after three.

In school children preliminary Schick and Moloney tests are done on the first visit. Next day the result of the Moloney test is recorded, and in five to seven days the result of the Schick test is read. In susceptibles we give 0·5 and 1·0 cubic centimetre at intervals of four weeks. This is also the procedure with the staffs of institutions.

The most important part of this work is undoubtedly that among infants and pre-school children. It is easier to organize a campaign in the schools, and most of our work has been done in school children. But increasing efforts are being made to protect the younger children over the period of greatest risk, which is obviously between the ages of one and six. Most of the infections and 70% of the deaths occur at these ages. It is also interesting to quote the opinion of Dr. Godfrey, of the New York Department of Health.

His observation is that the results of a preventive campaign depend chiefly on the success with which children of pre-school age are reached. He finds that even when 50% of children between the ages of five and fourteen have been immunized, the prevalence of diphtheria may remain substantially unaffected. (This does not mean that the immunized children are attacked, but refers to the rate among the general population.) If, however, in addition to the protection of half the school population, one-third of the children under five years is immunized, there is an immediate and sharp fall in the diphtheria rate. Over 1,200 pre-school children have been immunized in Melbourne without any trouble, and this work, on which we are now concentrating, is a safe and simple procedure.

Retests.

The retests after periods varying from three to twelve months, on children immunized by anatoxin have been very satisfactory. Among 500 school children previously susceptible, 88% showed no

reaction to the Schick test after two doses, and among infants over 90% were immune when they were tested after six months. Since I have been using three doses in infants this percentage has been increased to about 98%, only two or three modified reactions being shown in our series.

Immunization in Institutions.

It has been our practice to immunize the nurses at public hospitals for several years, and amongst several hundreds we have had only three infections in nurses who had been completely immunized. Several infections developed among nurses during their course of treatment, but there has been a satisfactory reduction in the number of cases in those institutions which have been visited and treated regularly.

One of the early institutions we treated was a home for infants. Several cases of diphtheria had occurred among the staff and infants, with one death, that of a baby of nine months. They were all immunized and retested in a month, when 90% were immune.

A little later, in sixteen of the immunized children positive nasal swabs were observed at various times. These were all very young children, but having demonstrated that they were non-reactors to the Schick test and should be protected, we isolated and watched them very carefully for further developments. With the courage of inexperience I withheld antitoxin, though it was available at any moment. Not one of these children showed any clinical signs of diphtheria, though two new nurses contracted diphtheria within a week of going on duty, so there was no doubt about the virulence of the germ. Later, these children went to a home in Bendigo, and the Medical Officer of Health, Dr. Kerr, reported that, although diphtheria was very prevalent in Bendigo and was several times introduced into the home, none of the 40 odd immunized children, then aged from two to four years, was affected.

If diphtheria occurs in an institution and if you have a reliable staff to report the first signs of disease, it is, on the whole, wiser to immunize the inmates actively at once, rather than to produce a transient passive immunity with antitoxin.

Allergy.

Despite the Moloney test, occasional local and general reactions, redness of the arm and, in rare cases, a rise of temperature have been noted, mostly in older children, but they have not been alarming.

Two cases of allergy occurred in 1931. One occurred in a child who was sensitive to the Moloney test, but, owing to a mistake in her responding to another name, she received a larger dose than was intended. Another much milder reaction occurred in a non-reactor to the sensitivity test. Both had been immunized with two doses the previous year, but this was not known at the time. This is an important point. They may, if they have been treated with anatoxin previously, develop sensitivity between the doses. In these two cases there was swelling of the lips and eyelids and an urticarial rash, but no more alarming symptoms developed. The first child, whose reaction occurred in less than half an hour, was given adrenaline, 0·5 cubic centimetre of a 1 in 1,000 solution; the other recovered without treatment. I consider it a wise precaution now always to carry adrenaline. It was a useful warning and has made me very cautious about carrying out reimmunization with full doses. I never give more than 0·5 cubic centimetre for either dose, and sometimes only one to children who have received anatoxin the previous year.

Comparison of Antigens.

Efforts are always being made to improve the immunizing material. Successful immunization obviously depends on a sufficient dose of a sufficiently powerful antigen. Against this we have to consider the likelihood of reactions. Toxin-antitoxin was a very safe material in spite of several accidents which were no fault of its own, so to speak, for instance, the accidental contamination that happened at Bundaberg, and dissociation of toxin and antitoxin after freezing, which caused some deaths in America. It was not, however, in most hands, as powerful an immunizing agent as anatoxin. An experiment carried out in America on nearly 500 children divided into comparable groups showed 95% of successful immunizations with anatoxin against 64% with toxin-antitoxin, and our own experience has tallied very closely with this.

Successful immunization with anatoxin depends on its antigenic power, and the laboratories are always trying to produce a material with higher and higher flocculating index. The possibility of causing violent reactions with these high value toxins has, however, always to be borne in mind, and a balance has to be struck between reactions on the one hand and comparative ineffectiveness on the other.

Dr. Adey, of the Commonwealth Serum Laboratories, was good enough to outline for me the method of preparation, and, briefly, the effort has been to produce a material of increasing flocculating index in a broth of decreased nitrogen concentration. This was designed to eliminate reactions and has been successful in doing so; but, curiously enough, the actual immunizing power of these later preparations, as measured by retesting with the Schick test, seems slightly less.

As all concerned are agreed that freedom from reaction is vital in any measure that is to achieve popular approval, it is considered preferable to give an extra dose of a bland material than to attempt to increase its effectiveness at the expense of its innocuousness. For this reason I have no hesitation now in recommending three doses of the present type of anatoxin, 0·5, 1·0 and 1·0 cubic centimetre, repeated at intervals of three to four weeks, as giving a high degree of immunity with safety and freedom from unpleasant accompaniments.

The interval of dosage is important, and Dr. Adey's findings have agreed with the original findings of Ramon: that a second dose given at an interval of less than three weeks does not increase

the titre of antitoxin appreciably, and that intervals of three to five weeks, and as a general rule about four weeks, give the most satisfactory results.

Other materials that have achieved some popularity abroad are "T.A.F." (toxin-antitoxin floccules) and "A.P.T." (alum reprecipitated toxoid). Both these have the disadvantage of considerable increase in the cost of production.

On the other hand, toxin-antitoxin floccules can be used in those sensitive to anatoxin; and also it would be highly desirable to produce immunity with one dose. In the preparation of alum reprecipitated toxoid a toxoid of very high flocculating index (20 and over) is used, and a method of "one-shot" immunization, as it is called, was described in a letter to *The British Medical Journal* last year by Dr. Ashworth Underwood.

However, at present our results with formol toxoid or anatoxin, in effectiveness and safety, seem to indicate that it is the material of choice.

An interesting question arises as to what would be the effect of immunization if the more toxic strain known now as the *gravis* form of the *Corynebacterium diphtheriæ* became common, as it has done in other parts of the world. Would ordinary immunization stand up to this test? Antitoxin at first apparently failed and had eventually to be used in doses hitherto considered unthinkable before it was of any use at all.

In 1927 reports came from Berlin that antitoxin was not having the effect that was expected and that a type of diphtheria with severe toxic symptoms had arisen which was very resistant to antitoxin. These features were very marked in Leeds and led to the work of Dr. Anderson, Dr. McLeod and others on the typing of the organism. Working with a special medium—blood agar prepared with slightly heated broth, sterilized by filtration and containing 0·04% of potassium tellurite — they were able to distinguish the three forms, *mitis*, *gravis* and an intermediate form, in 90% of cultures in eighteen to twenty-four hours. This medium is said to give with *Bacillus diphtheriæ gravis*, the organism associated with the gravest types of diphtheria, a picture so characteristic that it is not likely to be confused with anything else.

According to Dr. McLeod's account, the *gravis* form grows with a granular deposit and pellicle in broth, has flattened, lustreless colonies of irregular outline on the special medium, and actively ferments polysaccharides. It is associated with the extremely toxic, rapidly fatal form of the disease.

The *mitis* form grows with uniform turbidity in broth, has a convex, partly translucent and light-reflecting colony, and does not ferment polysaccharides. I have here a copy of Dr. McLeod's paper, with the illustrations, sent to Dr. Dale.

Then Commander Dudley, at the Greenwich School, showed that, although he had in 1932 a great prevalence of the *gravis* type of germ in carriers (the swabs were all typed), no cases of clinical diphtheria occurred. There were a few cases of technical diphtheria and a great increase in the carrier rate. His results, briefly summarized, showed that in the Naval Training School, where there was a changing population of boys, aggregating 1,000 at any given time, there had been in the years 1919 to 1927 385 cases of diphtheria, while in the five years following immunization 44 infections labelled diphtheria were recorded, of which only eight were clinically recognizable. Of these, six cases occurred in Schick reactors awaiting immunization.

He concluded that, though *gravis* is more invasive than *mitis* and may occasionally cause a trivial form of illness in Schick "negative" people, active immunization is a sound method of protection against recognizable clinical infection with the *gravis* bacilli.

Diphtheria in Immunized Children.

As indicated by Commander Dudley's conclusions, there is naturally great difference of opinion in deciding what constitutes clinical diphtheria. I have been very much indebted to Dr. Scholes and Dr. McLorinan, of the Fairfield Infectious Diseases Hospital, for their careful consideration of such of our patients as have come under their care. As Dr. Scholes points out, Schick "negatives" who are on the borderline, possessing just enough antitoxin to prevent reaction to the Schick test, may receive a massive infection which overcomes their immunity. Similarly, children who have been immunized do not always respond by producing sufficient antitoxin within a reasonable time and may also be attacked.

It is gratifying to record that such cases have been very rare. There has been no fatality in a "negative" or immunized child, and no case in a child retested after immunization and found to be protected.

Several cases have occurred among those incompletely immunized, but these are not a fair test. As far as I have been able to trace, only 12 cases have occurred in immunized individuals and about 10 in Schick "negative", and none of these cases was severe. These figures relate to over 4,000 children who are recorded in our card index as having received two doses of anatoxin.

Diphtheria is always with us and at any time may flare up in a region which has heretofore been only moderately attacked. The prevalence in Western Australia and New South Wales last year is an instance of this.

In Melbourne, where the work of Schick testing and immunization has gone on with one or two breaks since 1925, we have had an excellent opportunity of observing the results of the method. Although the work received a serious setback after the Bundaberg accident, the Melbourne City Council, who were pioneers in this important branch of preventive medicine, decided to go on with it. Though the numbers have been far below what they should have been, we feel that we have definitely reaped some benefit.

One event of interest was a State-wide epidemic of diphtheria during the years 1931, 1932 and 1933. We shared in this greatly increased prevalence and,

were it not for the protective work, might have been expected to have a greater proportion than other parts, as happened in the last epidemic in 1921, when our rate far exceeded that of the metropolis as a whole. For not only have we a large proportion of crowded areas, but we number amongst our population several institutions for children which have a very large annual turnover of inmates who are not ordinarily residents of the city and have never had an opportunity of receiving protective treatment.

In spite of these facts we have usually shown a rate below that for the metropolis as a whole and have been responsible for a diminishing proportion of the total cases—11% in 1930, 9% in 1931, 7·5% in 1932, and 8% in 1933 and 1934.

In the nine immediately adjacent municipalities, also in the central crowded areas, the rate was much higher than ours. In some it rose to 400 and 500 per 100,000, whereas 333 has been the maximum in the city.

As our birth rate was diminishing, we calculated the proportion of cases of diphtheria per 1,000 births in the city against the nine contiguous municipalities and found that our rate per 1,000 births was 16% lower than theirs. Considering our disadvantages and the fact that we have never achieved such a percentage of immunized children as is necessary to show a dramatic fall in the morbidity rate, these figures are an indfcation of the value of immunization, particularly that of the pre-school child, which was begun systematically in 1931.

A leading article in *The British Medical Journal* last year drew attention to the fact that diphtheria, an almost preventible disease, still attacks some 40,000 people every year in England and kills 2,000.

In Australia, proportionally, the figures are slightly more alarming. In 1933 there were nearly 15,000 cases in Australia, and in 1932, 15,500. Actually 400 to 500 deaths occur every year, and the death rate varies between six and seven per 100,000.

Some Practical Results Elsewhere.

The pioneer work of immunization was carried out in New York by Dr. Park and Dr. Zingher, first with toxin-antitoxin, an under-neutralized mixture that, in their hands, gave excellent results, and since 1932 with anatoxin. After the first success the campaign slackened, and in 1929 was begun an intensive campaign.

A recent table prepared by the public health authorities shows that since 1929 the annual number of deaths has been reduced from 700 in that year to 100 last year, and the mortality rate from 11·24 per 100,000 of population to 1·38. The number of cases in 1934 was the lowest on record.

Results in Toronto.

In Toronto a most vigorous campaign was carried on, one of advertisement, lectures and advice generally. In a population of 600,000 they immunized in 1933 nearly 6,000 school children, 500 pre-school children. Private physicians immunized 600 children, clinics immunized 900 children, and child health centres over 6,000 children.

In 1929 they had over 1,000 cases with 64 deaths. In 1933 there were only 56 cases with five deaths.

In 1934 no cases occurred at all in October and one in November.

The rate dropped from 168 per 100,000, with a mortality rate per 100,000 of 10·6, to a case rate of 9 per 100,000 and a mortality rate of 0·8.

No individual protected by toxoid died.

These figures are almost unbelievable and could not possibly be accounted for by the well known periodicity. The rate may rise a little, but if the immunization is kept up there is no reason why this satisfactory state of affairs should not continue. It is reported that the laboratories in Toronto have ceased to make antitoxin and the diphtheria wards are nearly all closed.

Conclusions.

I may conclude with the recommendations of the British Medical Research Council published in 1927:

> The Ministry is so fully satisfied in advocating this line of prevention that it does not consider it necessary to conduct any further detailed inquiries, but will continue to urge its adoption by local authorities. It is felt that the very favourable opinion formed as to the value of this method of prevention should be made more widely known.
>
> The evidence . . . is sufficient to justify the experienced opinions of all who have devoted much study to the problem and hold that the Schick test and immunization constitute one of the most notable advances in the sphere of preventive medicine.

Scarlet Fever.

Our experience in connexion with the control of scarlet fever has been confined to outbreaks in institutions. Among the general population the disease has not been sufficiently prevalent or severe to justify the recommendation of widespread preventive measures, and the actual methods are still in the experimental stage.

The Dick test was first used in Australia by Dr. Helen Kelsey, of the Fairfield Infectious Diseases Hospital, in 1926. Dr. Kelsey succeeded in isolating a toxin from a strain of streptococcus obtained at Fairfield, which she considered was more or less specific in producing the disease locally. Dr. Kelsey had observed that scarlet fever antitoxin from abroad was not successful in producing a Schulz-Charlton reaction in cases in which she had tried it here. This, as you know, is the result of an intracutaneous injection of a diluted antitoxin into the skin of a patient showing a rash presumably due to scarlet fever. An area of blanching, due to the local neutralization of the toxin, follows in a few hours in cases that are true scarlet fever. Working with an antitoxin produced at the Commonwealth Serum Laboratories from the Fairfield toxin, one of our first experiments was to demonstrate a Schulz-Charlton reaction in several cases of scarlet fever in an institution and, acting on this suggestion of Dr. Kelsey, all the work of testing and immunization so far carried out in Melbourne has been with her Fairfield strain of toxin.

Standardization.

The difficulty of working with the scarlet fever toxin is that each fresh batch has to be standardized anew on known human susceptibles. No laboratory animal has so far proved quite satisfactory, though white goats and, later, white rabbits have been used with some success.

The preliminary work of standardization is a long process. Briefly, different dilutions of the toxin are taken and tests are done on a number of individuals of different ages. A dilution that gives a satisfactory reaction in various groups at various ages is regarded roughly as a satisfactory working standard, and 0·1 cubic centimetre of this dilution is called a skin test dose.

In our first tests, fortunately performed on a number of resident medical officers at a city hospital, a curious incident occurred. We decided to use Dick, Dochez, two American strains, and Fairfield strain for comparison, and as Dr. Kelsey had decided that 1 in 1,000 was the standard dilution for her toxin, we decided to use this dilution, and two others, one twice as strong and the other half. By a misunderstanding the material supplied was ten times as strong as was intended. Each of the volunteers received three intradermal injections of each of the three strains of toxin. In the majority, even with this very strong mixture, no reaction was observed, but in the susceptibles several very marked reactions occurred. One resident medical officer, who was apparently very susceptible, had a temperature of 39·4° C. (103° F.), very injected throat, and a typical rash, thus inadvertently repeating the Dick's original experiment, which showed that the soluble toxin was the cause of the characteristic symptoms. He rapidly recovered after scarlet fever antitoxin, but this was a most unexpected reaction, considering that he could not have received more than 200 to 300 skin test doses, and Dr. Dick reports that 80,000 doses given inadvertently did not produce any ill-effects. Our toxin certainly seemed to be particularly powerful. After doing a large number of tests we arrived at what we considered was a reasonable standard. By the courtesy of Dr. Morgan, of the Commonwealth Serum Laboratories, we were able to compare this skin test dose with the standard skin test dose from Washington, and thus to verify our experiments, as the results were almost identical.

For our first 450 tests the results were:

Under five years, 50% reacted.
From five to ten years, 30% reacted.
From ten to fourteen years, 23% reacted.
Adults, 23% reacted.

At the same time Dr. Dick's warning that the incidence of natural immunity is related not so much to age as to contact must be remembered. In an overcrowded institution the incidence may be as low as 10%, while in rural or suburban populations it may reach 85%. This relationship has been well illustrated by our tests on probationer nurses, many of whom come from rural areas, and commonly show a very high percentage of susceptibles.

The Dick Test.

A suitable dilution of toxin having been obtained, 0·1 cubic centimetre is injected intradermally. This toxin, by the way, is always issued with antiseptic, and in various comparative tests we found that this did not affect the strength of the toxin if it was used soon after dilution. The result of the test, both in appearance and duration, is in marked contrast to that of Schick. It begins to appear in a few hours and reaches a maximum eighteen to twenty-four hours later. In typical cases it is frequently surrounded by a ring of blanching suggestive of "circumoral pallor", and it is probably due to some similar local constriction of the blood vessels. Dr. Dick warns against interpreting faint reactions as "negative". If they reach the size of one centimetre in any diameter they must be regarded as positive. The reaction is usually more an erythema than a definite injury, like the Schick test, and if in doubt I often pass my finger up and down over the arm and, as the blood flows back, the limits of the test may be plainly seen. A good light is necessary, too, as the flush may be difficult to see on a dark or mottled skin.

An important point about the control solution, a similar amount of which is introduced into the other arm, is that the toxin is very stable to heat and actually requires to be boiled for about three hours to render it inactive. I have sometimes had anomalous results due to the time of boiling being too short.

On the whole, I have had so few pseudo-reactions that I do not regard a control as very important. If similar reactions are seen on both arms, this is taken as a non-specific reaction and is regarded as negative; and they are occasionally seen in those who have had scarlet fever. A pseudo-reaction usually shows induration. Dr. Dick says this should not occur with a true Dick reaction, but we often notice it, and one can only conclude that toxins vary very greatly in their non-specific composition.

Active Immunization against Scarlet Fever.

Although Dick tests and passive immunization had been used to control outbreaks in institutions, we had no previous experience to guide us when we began in 1928 to use diluted toxin for active immunization. After the skin test dose had been established, it was possible to prepare dilutions of toxin containing a calculated number of skin test doses per cubic centimetre. This is the unit for immunization and we decided to begin very cautiously. The Doctors Dick gave very large doses of their toxin, up to 80,000 skin test doses, but we have never been able to give more than a small fraction of these doses. Our usual procedure is to give 100 skin test doses as a first dose, then, if there is no marked reaction, 200, 500 and 1,000 at intervals of five to seven days. Children stand it very well, and I had no complaints among a fairly large batch of toddlers, in one of the children's institutions which was threatened with an epidemic in a ward of about 50 children under six. The majority of them were sensitive and were

immunized, and only one mild case occurred in a recently immunized child. But even in these small amounts we have had a considerable percentage of reactions in adults, and in about one-third of the cases have had to give slightly reduced doses.

For these reasons we made inquiries about a streptococcal anatoxin or toxoid, but at that time reports showed that it was still more likely to give rise to reactions. However, reports since then from various sources indicate that it is to some form of toxoid that we may look in future for the most satisfactory results. Toxin-antitoxin was used with success by Dr. Park in New York, but not on an extensive scale.

An account of a preparation of a toxoid from the United States Public Health Reports, 1933, by Dr. M. V. Veldee, is of interest and may be briefly summarized. A method was elaborated for concentrating the toxin of the hæmolytic streptococcus, by which its toxin content was increased approximately fourfold without the total nitrogen being increased above that now present in ordinary unconcentrated toxin. By the action of formalin and storage at 37° C. this material was detoxicated, so that at the end of sixty days less than 0·5% of the skin-reacting factor remains. Single injections into susceptible rabbits indicated that the detoxicated product possessed antigenic powers, though detoxication does apparently destroy a portion of the antigen. Three doses were given to a number of children under fifteen years of age, without any reaction in the majority, and over 80% became immune in one month.

However, in our hands the toxin has given very good results. Twelve cases of scarlet fever have been reported in immunized nurses—all mild and uncomplicated. Of these, seven had not had a complete course and could only be expected to show a partial immunity. Considering that they all suffered a considerable amount of exposure, these few infections among nearly 1,000 nurses tested are very satisfactory. The number of infections in nurses was reduced from about forty to about ten annually.

The Reliability of the Dick Test.

In our series of nearly 1,500 tests, only one case of scarlet fever has been reported in a non-reactor. This was of an unusual type.

The nurse in question had had scarlet fever previously and gave no reaction to the Dick test. She later developed a sore throat, from which hæmolytic streptococci were isolated, and she was found to be suffering from albuminuria. There was no high temperature, no rash, and none of the ordinary signs of scarlet fever were present, but she was regarded by Dr. Scholes as definitely suffering from scarlet fever.

Immunization in Institutions.

So far, all our preventive work against scarlet fever has been carried out in institutions, and usually in response to an epidemic among the inmates, particularly during the years of increased prevalence, 1931, 1932 and 1933. Altogether nearly 1,000 tests have been performed in institutions and over 300 individuals have been immunized. In the early days attempts were made to control the spread of scarlet fever by passive immunization, and an experience at a home for mothers and babies will give an illustration of the working of both methods, passive and active, applied to the same epidemic.

At this home two cases of scarlet fever occurred among the expectant mothers, and one in a member of the nursing staff, and the authorities proposed to give scarlet fever antitoxin to the whole population, about 80 in number. As this material is very expensive, they were, however, glad to accept our offer to subject the whole personnel to the Dick test and to give antitoxin only to those who were susceptible. This procedure also gave us an opportunity to test a number of infants. Thirty-six infants were tested; seven were under six months, and of these only two reacted. Of the 29 over six months, 14 reacted and 15 did not react. The 16 reacting babies were given antitoxin. Of the staff and mothers, 40 were tested, and 11 out of 13 who reacted were also given passive immunization.

At the same time a hunt for carriers was instituted, and swabs and plates were taken from sore throats, nose and ear discharges. In this instance I obtained freshly made blood agar plates from the university and inoculated these directly the swab was taken, and took them straight to the laboratory for incubation. Although I do obtain satisfactory results with the dry swabs, I am sure the method of plating, where it can be carried out, is more satisfactory. Several children showed the presence of a hæmolytic streptococcus and were isolated and treated, and the majority cleared up. Two babies with persistent ear discharges had to remain in isolation for some weeks. No case occurred in the staff for four weeks. Then, owing probably to the fact that they had to take it in turns to attend to the "carrier" children, infections began to occur, and five of those nurses who had been shown to be susceptible and whose passive immunity had faded, became ill with scarlet fever. No individual shown by the Dick test to be immune developed the disease, though all were exposed. Three new untested probationers also contracted the disease, and we decided that, although immunity might develop more slowly with active immunization, it was the only rational procedure.

The whole adult staff was then retested and those reacting were immunized, and no probationer was accepted until she had been immunized. Of 39 adults, 13 were reactors and were immunized, and no other case occurred.

Similarly, at one of the hospitals a few cases occurred in connexion with one ward. A child with a mastoid gave hæmolytic streptococci on swabbing and was probably the source of the infection. Dick tests were done, so that only non-reactors should be allowed on duty in that ward. One nurse who reacted to the Dick test, and later two untested nurses and one child contracted scarlet fever, and cases began to appear in other parts of the hospital. The medical superintendent decided that the whole

staff should be tested, and 180 tests were done. Of the 85 reactors, 64 were immunized, and this hospital instituted the Dick test as a routine measure for all newcomers and has had only an occasional case since. Active immunity develops quickly and, if sufficient doses are given, this is the best means at our disposal at present for preventing the spread of the disease.

Our observation is worth recording on the effects of immunization on members of the nursing staffs. I invariably found, when I immunized the whole staff, many of whom had been several years in residence, that, although fewer of the older inhabitants were susceptible, they were much more likely to be upset. The probationers usually took the full course with the minimum of disturbance, but those who had been associated with the hospital for years very often had to have their doses reduced or even discontinued because of sore arms.

For this reason, and also because they are at their maximum susceptibility, we advise the hospitals to have the work done during the preliminary weeks of training.

Outbreak of Scarlet Fever with Glandular Fever.

In May, 1933, we had a rather curious outbreak in a home for children. A case of scarlet fever was reported, and when we visited the home we found that two children who had been very ill two weeks previously with what appeared at the time to be mumps, were showing signs of desquamation. At the same time there had been a widespread prevalence of sore throats and swollen glands, and of the 54 inmates nine had shown symptoms of severe illness. Of the nine patients, all had fever, swollen glands, and sore throat, and in some cases vomiting in the early stages. No rash had been observed in any of these cases. Both staff and inmates were submitted to the Dick test; but two more had developed scarlet fever before the test was done.

Of the 51 tests, only nine reacted to the Dick test. Thirty-four of the children were under ten years of age, and of these only five reacted. This is, of course, an extraordinarily low figure and indicated that the harbouring of the germ had been almost universal and that most of the children had harboured the germ without showing definite symptoms of the disease. The connexion with the swollen glands seemed to be accidental, because one child who had marked cervical glands without any raised temperature or malaise reacted to the Dick test, and later two other children developed swollen cervical and inguinal glands without a rise in temperature.

I was interested to read later in *The British Medical Journal* an account by Dr. Bradley, surgeon of Margate District Hospital, of an outbreak of glandular fever in a school. In this case there were 11 children out of 84 who showed severe, and in two cases alarming, attacks of glandular fever, and although in this case none seemed to have developed scarlet fever, the medical officer found that "sporadic cases of glandular fever had arisen in the district during the previous two months which had been prolific in producing other throat infections and notably scarlet fever".

In our case the glandular condition did not seem in itself to give rise to fever and malaise, but the application of the Dick test was of considerable interest in showing that evidently two organisms were concerned: one the hæmolytic streptococcus, which was found on swabs of several who had recovered; and the other some organism probably similar to that concerned in the outbreak described by Dr. Bradley, but of much less virulence.

Possible Relationships with Other Streptococci.

Many authorities doubt whether the scarlatinal streptococcus and the tests connected with it are as specific as, for instance, the diphtheria bacillus and the Schick test.

Dr. Pickering Pick, for instance, in a report on the epidemiology of scarlet fever, suggested that many forms of streptococcus, usually widespread, throughout the community, causing sore throats, erysipelas and allied conditions, give some immunity to scarlet fever and that it is only when for some reason streptococci have been scarce that epidemics of scarlet fever occur.

My own experience is so limited that any observations I may make are put forward only tentatively. I tried to establish a relationship between the occurrence of septic fingers and the reaction to the Dick test, because I had been struck by the fact that several nurses developed septic fingers while under treatment, and in every case they were Dick reactors.

In the only hospital where the Dick test has been used as a routine procedure, I also made inquiries as to the incidence of septic fingers, but at the time no records were available. I was interested recently, therefore, to hear from two senior sisters who had gone to positions there from other hospitals, that they had never seen such a low incidence of septic fingers in any hospital.

Again, the relationship between cases of scarlet fever and puerperal sepsis, as seen in infection by carriers—the treatment of puerperal sepsis, erysipelas and other septic infections with scarlatinal antitoxin—do not these instances point to some common factor in the exotoxins of apparently different strains of streptococcus? And is antitoxin proved to be effective in such cases?

On the other hand, the occurrence in convalescence of relapses and complications, considered to be due to strains other than that causing the original scarlet fever, suggests rather a limited than a wide embracing valency, and would suggest that antitoxin against scarlet fever is not effective even against all the forms of this disease, much less against a wide series of allied conditions.

A large series of Dick tests on expectant mothers would probably show whether immunity against scarlet fever is any indication of protection against other forms of streptococcal invasion. However, I

merely suggest this aspect to invite discussion from those more qualified than I am to speak on the subject.

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THE HYPOCHLORITES AS ANTISEPTICS.

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As the basis of the treatment of septic wounds rests more or less on the reliability of the antiseptics we use, perhaps our experience of one group of them—the hypochlorites—would be of interest. In the experience of one of us (H.S.S.), who has had a wide experience of wound treatment, military as well as civil, the hypochlorites have proved themselves the most efficient (and inexpensive) antiseptics in use today. These substances have long been known as antiseptics. Their history is discussed by Dakin.[2]

Of course we assume that every surgeon realizes the important factors governing the use of antiseptics, namely: (i) that they shall be innocuous to the tissues on which they are applied; (ii) that it is futile to expect them to act by merely pouring them on the wound—they must be in contact for a sufficient time; (iii) that the lotion must be of accurate concentration, which in the case of the hypochlorites under discussion should be approximately 0·5% of sodium hypochlorite or its equivalent.

Because of the burning pain on application, the hypochlorite solution is diluted by some surgeons, for example, 1 in 4, surely showing a lack of knowledge of the principles underlying the whole treatment. They are correcting the excessive alkalinity of an improperly made solution at the expense of the hypochlorite antiseptic content, a procedure they repeat continually, regardless of the care which may have been taken with the preparation of the solution.

Long experience has shown the efficiency of these various hypochlorite solutions, but it has also disclosed some drawbacks. The first of these was the presence of free caustic alkali, such as one gets in the commercial hypochlorites. Very many years ago *Liquor Sodæ Chlorinatæ* (Labarraque's solution) was in surgical use, but, owing to its irritating qualities, was abandoned. As a matter of fact, the proportion of alkali contained in this solution is great enough to produce solution of the skin, if the contact be sufficiently prolonged. It has also been clearly shown that the bactericidal quality of hypochlorite solution is not due to its alkalinity.

The first hypochlorite solution to be used in the Great War was eusol, introduced by Lorrain Smith (1915). In this, 12·5 grammes of bleaching powder are shaken vigorously in one litre of water, then 12·5 grammes of boric acid are added; the mixture is shaken and allowed to stand overnight; the clear fluid decanted off is ready for use next morning. Hypochlorous acid in the strength of 0·54% is the antiseptic agent.

Next came Dakin's (1915)[1] solution—a neutral hypochlorite solution in which 400 grammes of crystallized sodium carbonate are dissolved in 10 litres of ordinary water, and 200 grammes of good quality chlorinated lime are added to it; the mixture is well shaken; in 30 minutes the clear fluid is siphoned off and filtered through cotton wool. To the filtrate are added 40 grammes of boric acid; the solution may be used at once. The mode of preparation is simple, but the compact masses are often incompletely broken up and the boric acid contained in it often seems to cause irritation.

Then came Dakin's solution, prepared by Daufresne's method, in which chlorinated lime (having 25% active chlorine), 184 grammes, sodium carbonate (anhydrous), 92 grammes, and sodium bicarbonate, 76 grammes, are dissolved in 10 litres of water. The solution should contain 0·475% of sodium hypochlorite. The drawback is the variability in the content of active chlorine in the chlorinated lime.

The excess of caustic alkali which constitutes the irritating element most to be dreaded in hypochlorite solutions is neutralized by boric acid in the preparation given by Dakin; it is not present in Daufresne's preparation.

Conflicting reports as to the value of hypochlorite solutions from various local sources, and occasional unsatisfactory results obtained by one of us (H.S.S.), in spite of uniformly satisfactory experience with these solutions in other parts of the world, led to the chemical investigation of a number of these solutions. The specimens were obtained more especially from places where unsatisfactory reports had been received.

The determination of the available chlorine or sodium hypochlorite content of these solutions is very simple. Excess of potassium iodide is added to a measured volume of the solution. This is acidified with acetic acid and titrated with N/10 sodium thiosulphate solution to the disappearance of the iodine colour. The addition of a starch solution as an indicator is not necessary. If the solution is in the vicinity of the concentration of sodium hypochlorite which it should contain (0·5%), five millilitres is a convenient volume for titration. This is diluted with about three volumes of water before titration. The present results have been expressed as grammes of sodium hypochlorite in 100 millilitres

of solution (1 millilitre of N/10 sodium thiosulphate = 0·00745 gramme of sodium hypochlorite or 0·00355 gramme of available chlorine).

The investigation began with Dakin's solution, but owing to the almost incredible variation found among samples it was extended to samples of other types of hypochlorite solutions. Figures obtained in the different solutions are given in Table I.

TABLE I.

Variation of Sodium Hypochlorite Content (Grammes per Hundred Millilitres) Hypochlorite Solutions from Various Sources.

Dakin's Solution.	Eusol.	"Calsol."
0·27	0·01	0·26
0·72	0·40	4·22*
0·88	0·57	0·58
0·94	0·78	0·55
0·43	0·74	0·98
0·13	0·04	0·45
0·35		0·59
0·87		0·62
1·43*		0·62
0·55		0·52
0·56		
0·91		
0·41		
1·60*		
0·17		

Excessive variations of the concentration of Dakin's solution observed in Sydney Hospital were found to be due to variations of available chlorine content of the chlorinated lime used. Since a better quality product has been used and its available chlorine content has been determined before the solution is made up, variations of sodium hypochlorite content outside of tolerable limits (0·4% to 0·6%) have disappeared and no unsatisfactory results have been observed in the clinical uses of the material.

The occurrence among these figures of values considerably above the standard concentrations of 0·4 to 0·6 gramme of sodium hypochlorite per 100 millilitres (specimens marked with an asterisk in Table I) is due to the practice of some dispensers and manufacturers of making these solutions up in bulk at high concentration with a view to increasing their stability. In the light of our experience it is a practice strongly to be condemned, unless sufficiently explicit directions for diluting are supplied and noted on the label.

The occurrence of solutions which caused irritation among those examined is thus readily explained. The occurrence of solutions too weak to be effective remains to be considered. Most hypochlorite solutions are not stable, reacting apparently with the carbon dioxide of the air. With chlorinated lime, for example, $CaClOCl + CO_2 = CaCO_3 + Cl_2$.

A few experiments were carried out to determine the rate at which some of these solutions decomposed under different conditions of temperature and illumination.

The figures in Table II indicate that "Calsol" is superior to the other solutions examined in its stability at temperatures up to 37° C. This solution keeps practically unchanged for over a fortnight at room temperature, and for a week at 37° C. At the end of a month at room temperature, however, its concentration of sodium hypochlorite has fallen

TABLE II.

Effect of Temperature on Stability of Hypochlorite Solutions.

Concentrations as grammes of sodium hypochlorite per hundred millilitres.

Solution.	Sodium Hypochlorite Percentage.		Period Exposed.	Temperature.
	Initial.	Subsequent.		
Eusol	0·04	0·03	7 days.	Room.
	0·78	0·50	7 days.	37° C.
	0·74	0·76	1 minute.	Boiling.
Dakin's	1·60	1·59	2 hours.	Room.
	1·60	1·55	3 hours.	Room.
	1·60	1·55	11 hours.	Room.
	0·41	0·13	7 days.	37° C.
"Calsol"	0·45	0·45	15 days.	Room.
	0·50	0·55	18 days.	Room.
	0·55	0·34	32 days.	Room.
	0·52	0·55	7 days.	37° C.
	0·53	0·50	19 hours.	37° C.

materially. The figures in this table show that Dakin's solution keeps better at a higher concentration than at that at which it ought to be used. The table also shows that rapidly raising certain of these solutions to the boiling point and then allowing them to cool does not appreciably decompose the hypochlorite present. On the contrary, there is an increase of concentration owing to the evaporation of water.

The effect of exposure to diffused daylight was observed on solutions of "Calsol" at room temperature. The results are shown in Table III.

TABLE III.

Effect of Light at Room Temperature on the Stability of "Calsol" Solution.

Concentrations as grammes of sodium hypochlorite per hundred millilitres.

Sodium Hypochlorite Percentage.		Period Exposed.	Illumination.
Initial.	Subsequent.		
		Days.	
0·58	0·55	18	Light.
0·58	0·57	18	Dark.
0·55	0·34	32	Light.
0·55	0·54	32	Dark.
0·55	0·33	42	Light.
0·55	0·53	42	Dark.

Up to a period of eighteen days light made no appreciable difference to the stability of the solution. Solutions which had decomposed considerably in a period of thirty-two days when exposed to light, remained practically unchanged when kept in the dark up to forty-two days.

It should be remembered that "Calsol", as bought, must be diluted with nine volumes of water before being dispensed.

The relative cost of twenty ounces of the solutions discussed, in the concentrations used on the patient, are as follows: eusol, one penny; Dakin's solution, one penny; "Calsol", five pence halfpenny.

We are indebted to Mr. Kerr, M.P.S., Chief Dispenser at Sydney Hospital, for this information, which applies to the purchase of the material in bulk.

Summary.

Considerable variations were observed in the efficacy and hypochlorite content of Dakin's solution and eusol.

Solutions of "Calsol" showed very little loss of strength after exposure to light at room temperature for eighteen days, but at the end of thirty-two days considerable loss had occurred.

No loss of strength occurred during exposure to room temperature for forty-two days, to 37° C. for seven days, or to 57° C. for nineteen hours in the dark.

Bringing to the boil does not materially alter the concentration of "Calsol" or Dakin's solution.

Dakin's solution and eusol do not stand exposure to light as well as "Calsol".

It is wise to adhere in clinical practice to well proved hypochlorites; some brands on the market are valueless.

The correct strength is the beginning and end of the whole matter. To dilute properly made hypochlorite solution is to spoil it as an antiseptic.

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SOME ASPECTS OF THE MEDICAL CURRICULUM OF THE UNIVERSITY OF SYDNEY THROUGH THE EYES OF A RECENT GRADUATE.

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A MEDICAL curriculum is a debatable thing. In undergraduate days there is a strong temptation, not often resisted, to criticize various aspects of the course. Many a time the basis of this criticism lies in the feeling that too much time is allocated to this subject, insufficient to another. Quite often in weighing considerations such as these, the time factor and practical value, when thrown into one scale pan, are not nicely balanced by the academic side, and it is with this particular point that not only undergraduates, but also graduates and those who are empowered to arrange the curriculum, are concerned.

After graduation men are rarely tempted to criticize remediable deficiencies of the course simply because they find it easy to adopt an attitude of laissez-faire. "We are through, so why should we worry", they say, not always in effect alone. As a result, those who control the training of students in the faculty are deprived of a valuable source of criticism, which, based on accumulated experiences and observations and incorporating constructive ideas, could be of great assistance to departmental administrators. It is convenient at this point to examine the question of a satisfactory course.

According to Professor C. G. Lambie and Professor Harold R. Dew,(1) "the medical curriculum should be framed with a view to securing not only an adequate professional training, but an academic and cultural background capable of providing the means of growth and development". Many will be in accord with this conception. Some, however, will advocate the introduction of more practice to replace a deal of the purely academic, others will contend that little departure from the present arrangements is warranted, while there is a third school which insists with John Newman that a university "contemplates neither moral impression nor mechanical production; it professes to exercise the mind neither in art nor in duty . . . it educates the intellect to reason well in all matters, to reach out towards truth, and to grasp it".

Whatever the merits of these conceptions, it is an obvious fact that the Sydney curriculum is being subjected to long overdue changes and revisions, which show that the authorities appreciate their responsibilities to medicine, to the University of Sydney and to the all-important *alumnus*. More changes and revisions are necessary, deletions and additions, too. The Medical School of the University of Sydney has a proud position to maintain and to consolidate, for in a report submitted to the University of Oxford recently it was stated that our university was preeminent of the Empire schools in teaching and examinations.

Now, in an arbitrary sense, the medical course is limited to six years. In that, the minimal time, an undergraduate may successfully scale the academic Mont Blanc, and well may he shout "Excelsior!" as he looks back to survey exultantly the steep ascent, the by-paths beset with difficulty, and the thin ice of examinations over which he skated in November (and perhaps in March). Momentarily he may feel secure, that his days of study are over, but soon he realizes that he will be a student all his life, that a medical course has length, breadth and height, and that a succession of peaks of even greater altitude lies ahead to test his mettle.

To prepare the climber for these tasks, the university course should be a model of thoughtful planning. The aim should be to provide modern approach to the individual subjects, keeping the goal of general medicine in clear view and maintaining as broad an outlook as is compatible with that objective.

At once it is pertinent to focus attention on matriculation. The wisdom of the step of including physics and chemistry as portion of the requirement for entry into the Faculty of Medicine, is winning favour. This would lessen tedious and unnecessary revision for the greater proportion of students. Courses lasting one term each would be sufficient, and would cater for those who had not reached the higher standard. Radical changes are indicated with regard to organic chemistry. Instead of loading the student mind with needless detail on dyestuffs and matters of interest only to an organic chemist, the

course should have a strong medical flavour, designed as an introduction to biological chemistry, and delivered by a man in sympathy with medical requirements. The anomalous position whereby students are required to cram an important subject (judging by the percentage of failures) in the last term of the year calls for immediate rectification. Botany and zoology as introductory sciences are in correct proportion with one and two terms respectively, but overlapping should not be permitted. Additionally, zoology could be shaded into anatomy, embryology and physiology in the third term.

In second year anatomy should be taught. The practice of giving three manuals and a cadaver to a student, and expecting him to learn anatomy, and to remember it, can be paralleled by the ancient and effete device of teaching swimming by pushing a learner into a pond with the advice: "Swim!" Good and sufficient teaching encourages students to appreciate dissections. More demonstrations and supervision in the dissecting room are necessary, and their absence will serve to maintain the present state of knowledge, which at best is "book knowledge", and which cannot apparently be retained after the third year examination, as Professor Dew and other surgical tutors are aware. Arouse the interest of the men in their work, let precept be reinforced by example and the student's knowledge of anatomy will grow.

It is more difficult to legislate for physiology with its less accurate detail. In view of the excellent text book available, the need for such a vast amount of lectures with the outward limbs and flourishes is barely apparent when compared with the twin fundamental, anatomy. The system of cyclostyled lecture notes is good, but would be more effective, perhaps, if the notes were made available the night before the lecture. There should be a greater correlation, too, between lecture material and subsequent clinical requirement, and it is a pity that the opportunity is neglected of illustrating departures from the normal by reference to the abnormal. In the present state of affairs, students enter the hospitals almost prepared to meet laboratory animals instead of patients suffering from disordered physiology. Neurology would become more intelligible as well were clinical examples of lesions exhibited. Surely the position can be met by instructive reference to applied physiology and more expert sifting of the wheat from the chaff.

Interests sharpen when the second half of the course is commenced. The student is now to be equipped for the work of general practice, which is the ultimate aim of 90%.

In this respect the "attempt to introduce the various subjects of medical science in a rational sequence and to correlate them with one another in such a manner as to give a due sense of their essential unity and inter-relationship"[1] represents an advancement in medical teaching. Full effect should be given to the ambitious plan of attack laid down. Bury the system of water-tight compartments into which the subjects were made to fit under the old *régime*.

Pathology and bacteriology are stimulating. Yet an excellent opportunity is lost in "path." to consider the important relation of lesions to symptoms and signs. That is what the student requires to know. Bacteriology, as taught by Professor Wright, was splendidly useful, and promoted a spirit of critical inquiry. Still some of the staining methods could not be carried out at the hospitals where the requisite materials were lacking. Pharmacology is a hardship—'twas ever thus. Only a keen teacher, systematic lecturing, condensation and elimination of unnecessary detail and the provision of typed notes *pari passu* with lectures offer any hope of making it a useful part of medicine and affording help to "the already overtaxed student who often fails to get any idea of the subject at all if the matter be not placed in a condensed form within his reach" (Sir William Whitla). Certainly the futility of providing one-sixth of the lecture notes in "stew-vac"—an actual happening—is unmistakably apparent. Of therapeutics it can be said that the present arrangements are not good. It might be better to give a very brief summary of the principles of treatment to round off each lecture in medicine and surgery. These could be complementary to the routine lectures for which the notes should be immediately available at the lectures. In any case most of the treatment of disease is learned in actual hospital practice, but this raises another issue, which will be disposed of later.

Before medicine, surgery and obstetrics are considered, reference must be made to the specialties. Psychiatry is fairly well taught, though in incorrect proportion to the other specialized fields. Normal psychology and disorders of children warrant closer attention. Demonstration-lecturettes, the eye, ear and hand cooperating, are the keynote in successful teaching and assimilation. This point of view can be substantiated by any student who has attended for "skin" demonstrations in the "glass house" at Royal Prince Alfred Hospital. The coordination between patient, teacher and student was admirable, and the scheme is worthy of emulation by other departments.

Weary mountaineers are enabled to take a welcome respite in preventive medicine, medical jurisprudence and medical ethics. If examinations are necessary, they should be conducted at the end of Trinity term or in the first week of Michaelmas term. Thus a clear twelve months could be devoted entirely to the major subjects of the curriculum and preparation for the examinations.

What of obstetrics? Here we meet a compact subject convincingly taught and demonstrated. Lectures, however, should be approximated more closely to the period of residence at hospital. If given at the end of fourth year, with examinations in pathology, bacteriology, pharmacology and *materia medica* hard by, students cannot be expected to pay more than scant attention to obstetric

lectures. Concerning the actual residency in hospital, two faults may be mentioned. There is a limit of cases at the Royal Hospital for Women at which students may "assist". Contradistinctly, nurses obtain more "assists" than a student—an amazing incongruity which should be removed. Secondly, students are not allowed to conduct a vaginal examination during labour, though this examination is permitted for nurses.

Next to be discussed in this brief survey are surgery and medicine. In fourth year students are keen to grasp any opportunity to gain knowledge, practical and theoretical, of sufferers and suffering. Tutors should be prepared to accept the fact that a lot depends on their efforts. Keenness breeds keenness. Punctuality on both sides would be beneficial. Laxity in this respect will act as a brake on keenness and, moreover, will conduce toward hastily secured histories, too rapid observations and physical examinations and, possibly, faulty processes of reasoning. Consciously or unconsciously, students model their methods on those that they see employed commonly; hence the need for the highest possible standard of teaching. On the surgical side, elements of surgical practice, first aid, bandaging, suturing and dressings should be considered, while medical tutors have as their objective the task of making students familiar with signs and symptoms and methods of examination. Here a standard teaching is necessary, and even though Professor Lambie's scheme or clinical text books insist on minutiæ, detailed work will lay a dependable foundation. Haphazard methods and the bland assurance "when your judgement is good, give no reasons" are not calculated to develop efficient practitioners. At the out-patient departments methods of teaching vary. Superior to any other scheme is that which provides for a thorough investigation of half a dozen selected cases, with a rapid overhaul of the remainder. It is surprising to find how few tutors adopt this commendable plan.

Ward work can be most instructive. Invariably fourth year students are keen. Much can be taught, much can be learned. Let the students be seated at the bedside if a case is to be considered at length. Why must they stand all the afternoon? Let there be vigorous teaching. Question, demanding answers. Salt the commonly occurring conditions with the recondite. Arouse interest and maintain it.

Fifth year affords greater opportunity for ward work, though two terms are spent away from the hospital, one at the Royal Alexandra Hospital for Children, the other at the obstetric hospitals. Professorial clinics have proved excellent in consolidating foundations laid earlier. Here there are special opportunities to combine theory and practice. Especially is this so on the surgical side. Students from all teaching hospitals should be able to share in the full benefits to be derived from professorial direction of activities. In the term's work with the professors of surgery and medicine, biochemical, bacteriological and pathological tests can be carried out under optimum conditions which contrast sharply with those obtaining in the general wards, where minor obstructions, for example, poorly equipped laboratories, disorganize what should be smoothly running machinery.

Having entered into his sixth year, the student is confronted with a difficult problem. Looming large is the examination spectre, and failure in the final incurs a drastic penalty, immediate and remote. So long as knowledge is measured by an academic yardstick there can be but one, a justifiable, aim on the student's part. Consequently he will cram himself with book knowledge, taxing both mental and physical powers. He knows that in the final the opportunities to present the practical viewpoint are negligible. Further, it is a moot point whether such a time is opportune to discuss this aspect of medicine. In these circumstances students are driven to their books, often when their love of medicine dictates the assimilation of useful details of hospital practice, which corresponds with general practice.

Before a partial solution of the difficulty is suggested, some other issues should be reviewed. Take surgical anatomy. Sixty students, two cadavers—absolutely futile! Again, anæsthetics; these are very important. But why have to wait in the theatres for a whole afternoon for one anæsthetic? In regard to fracture work, students want to see fractures reduced and "put up", and opportunities are rare. Sets of representative skiagrams should be available at all times so that students may develop proficiency in their interpretation. Finally, how many students see patients with acute medical and surgical conditions immediately on admission and before therapeutic measures have been adopted? Very few. The remedy is obvious.

The introduction of a short period of residency in hospital in final year would be an invaluable innovation. In one month a student working under supervision could acquire a great deal of useful first hand knowledge of medical and surgical emergencies and their treatment. This system works admirably in obstetrics and it should be adopted for surgery and medicine, to bring about a harmonious blending of the academic and practical, and to fit the men for their term of office as junior resident medical officers.

In closing this contribution it is barely necessary to state that an exhaustive analysis is impossible. Should administrators require points of perspective, let them turn to the recent graduate and undergraduate. The onlooker is credited with seeing most of the game. The player, however, has a viewpoint which is to be considered. Should the onlooker be a professor, he should invite and be tolerant of constructive criticism. It takes students as well as professors to make a university.

There will always be clashes of opinion regarding examinations, their worth and their scope of usefulness. Yet on one point there need be no argument.

When a student fails in an examination, the teacher should regard it as his own failure to instil the principles, until he can establish evidence to the contrary. Students should be entitled and encouraged to discuss their failings freely with

teachers. Their very discussion will remove inhibitions, provide for the cultivation of a more pleasing relationship between examiner and examinee, and help to raise the standard of knowledge more effectively than can ever be expected from the piling on of extra work and the mere elevation of the pass mark.

Reference.

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Reports of Cases.

NEURO-SARCOMA OF THE MEDIAN NERVE.

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Clinical History.

MR. L.W., aged thirty-eight years, consulted me in July, 1934. His complaint was that he first noticed "numbness" of the tips of the index and second fingers of the right hand two years ago and that when he bumped the inner side of the right arm above the elbow he felt pain shoot down the medial side of the arm to the area of "numbness". This persisted till four months ago, when he first noticed a lump about the size of his thumb nail on the inner side of his arm, 10·0 centimetres (four inches) above the elbow. He noticed also that pressure over this lump caused the shooting pain down the arm. The lump had grown steadily to its present size, and during the past few weeks the pain had become constant, very severe, and "surging" in character.

On examination a fusiform swelling the size of a small hen's egg was palpated on the medial side of the right arm, 10 centimetres above the medial epicondyle. It lay in the bicipital groove, was smooth and rounded in contour, tender on pressure, and firm, but not hard, in consistency. The tumour was not attached to the overlying skin, was easily moved laterally, but not longitudinally. The axillary glands were not palpable, and there was no impairment of function, power, coordination or sensation in the arm or hand. The general examination revealed nothing further abnormal, except the presence of advanced spondylitis and osteoarthritis of the pelvic and hip joints. A provisional diagnosis of neuro-fibroma of the median nerve was made.

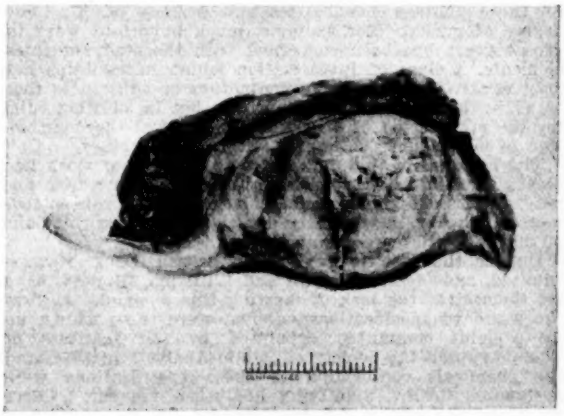
At operation the tumour was exposed by a longitudinal incision over it; the overlying tissues were vascular, the tumour was semi-fluctuant, easily isolated from the surrounding tissues, and was found lying in the trunk of the median nerve with the nerve fibres spread out over it. The tumour, together with 6·75 centimetres (two and a half inches) of the median nerve above and below it, was removed intact and the wound was closed. When cut into, the tumour was soft, greyish yellow in colour, and oozed fluid. The pathological examination showed the tumour to be a neuro-sarcoma measuring 4·7 by 2·8 centimetres, spindle shaped, parts of it being cellular and parts necrotic. The tumour was malignant, the degree of malignancy being uncertain. No evidence of newgrowth was detected in the proximal or distal segments of the nerve. At the time of writing, which is six months after the operation, the patient has a useful arm, devoid of pain, his main trouble now being the coldness of the tips of the index and second fingers and the loss of flexion at the interphalangeal joint of the thumb. Other demonstrable findings of the median nerve paralysis noted in this case are: wasting of the muscles of the volar-ulnar aspect of the right forearm, impairment of pronation, some loss of power in the forearm, impairment of abduction of the thumb, considerable loss of flexion at the distal interphalangeal joint of the index and second fingers. There is some loss of epicritic sensibility over the radial half of the palm, the flexor surface of the thumb, and the proximal halves of the index and second fingers and the radial side of the ring finger. There is some loss of protopathic sensibility over the volar and dorsal aspects of the distal halves of the first and second fingers, together with disturbance of deep sensibility and trophic changes affecting only the distal phalanx of the index finger.

Comment.

Interest is attached to the case for the following reasons:

1. The infrequent occurrence of solitary neurogenic sarcoma in general practice.
2. The patient now affords an opportunity for a study of the effects of complete division of the median nerve above the elbow.
3. There were no other detectable tumours of nerves nor any stigmata of von Recklinghausen's disease, the tumour being solitary.
4. The literature on the subject of neurogenic sarcoma shows that considerable difficulties have existed and still exist as to the correct nomenclature and histogenesis of these tumours.

As to the origin of these tumours, the literature indicates that there are two main schools of thought. Penfield[1] and Mallory[2] and other American pathologists believe they arise from the supporting endoneural connective tissue and are therefore mesodermal in origin. Verocay[3] and Masson[4] and others, on the other hand, consider that some, if not all, of these tumours, arise from the Schwann cells of the nerve sheath (migrants from the neural crest) and therefore are ectodermal in origin.



Photograph of the tumour sectioned longitudinally, showing it to be spindle shaped and well encapsulated, and showing the absence of infiltration.

The prognosis, according to the literature, is bad. Few patients live longer than five years after the initial operation, metastases developing via the blood stream rather than the lymphatic, either under the scar or in some other part of the same nerve trunk (more usually the proximal segment), and eventually in the lungs. In a paper, entitled "Sarcoma of Peripheral Nerves", read by Professor Harold R. Dew at the meeting of the Royal Australasian College of Surgeons held recently in Melbourne, the author points out that "this tumour is a very treacherous one—of varying degrees of malignancy, but extremely prone to recurrence and to finally cause death from pulmonary metastases". He considers operation, if performed, should be radical, and "if recurrence, which is very common, takes place, amputation holds out the only chance of saving life".

It is interesting to note that at present (six months after operation) a small lump is palpable over the cut end of the proximal segment of the median nerve in the case here reported. This may be a stump neuroma or an early recurrence of the sarcoma.

It would appear from the literature that neurogenic sarcoma is radio-resistant, deep X ray therapy having been disappointing in its results, but nevertheless it is recommended post-operatively with the object of limiting recurrence.

Acknowledgements.

I wish to thank Professor H. R. Dew for his advice in this case, and also for giving me access to his paper, "Sarcoma of Peripheral Nerves". I also wish to thank Dr. George Bell for allowing me access to the hospital notes of the operation which he performed upon this patient.

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Reviews.

STANDARDS FOR LABORATORY CHEMICALS.

THERE is an ever-increasing necessity in scientific work for those who use chemical reagents to know exactly their degree of purity. For example, much important work in recent years has been concerned with the study of trace elements, a class of investigation which necessitates not only relatively accurate determination of substances that occur in the most minute quantities, but in addition calls for the preparation of experimental material the composition of which must be known with great accuracy.

The progress of this and many other types of work has created an increasing demand for chemicals which not only possess a high degree of purity, but, and this is still more important, in which the actual character and amount of the impurities present are known.

During the last twenty years British Drug Houses, Limited, and Messrs. Hopkin and Williams, Limited, have set themselves the task of meeting this demand. At first the standard specifications adopted were those drawn up by a joint committee appointed by the Institute of Chemistry and the Society of Public Analysts in 1914, and the chemicals conforming to these specifications were designated "A.R.", signifying analytical reagent. There were originally 88 "A.R." specifications, but the number of chemicals for which such specifications were desirable grew rapidly. The original joint committee, which had regarded its work purely as a war emergency measure, did not wish to carry its operations further and consequently British Drug Houses and Hopkin and Williams each proceeded with the production of their own specifications and chemicals, publishing separate collections of "A.R." specifications in book form.

Hopkin and Williams's most recent publication (1931), which was prepared and issued in collaboration with Baird and Tatlock (London), Limited, contained 178 monographs, and that of British Drug Houses (1932) contained 208 monographs. Although similar, the specifications were not identical, and the two firms wisely decided that confusion and misunderstanding would be avoided if a common set of standards were adopted. The outcome of the decision was the publication of the book of "Analar Standards for Laboratory Chemicals".[1]

The work contains 220 specifications to which the corresponding "Analar" grade chemicals made by either British Drug Houses or Hopkin and Williams are guaranteed to conform. The monograph for each chemical gives formula, molecular weight, list of impurities showing maximum limits for each, details of limit tests for each impurity, where possible a method of assay, and in some cases physical constants.

The tests are well selected and following the practice adopted in the British Pharmacopœia, 1932, framed so as to eliminate ambiguity. For example, whereas in the old "A.R." standards one might find the statement that in the test for chlorides there should be "not more than a faint opalescence", or in the tests for heavy metals "not more than a faint darkening", in the "Analar" standards the statement is "any opalescence produced should not exceed that given by . . ." or "any colour produced should not be deeper than . . ." The appendix contains details of standard comparison solutions for opalescence, turbidity and colours for heavy metals and iron and phosphate and silicate.

The laboratory worker will find the book a convenient one to consult for many everyday purposes. It is a record, for example, by those best qualified to know, the manufacturers, of the impurities to be expected in the various chemicals, and this information is frequently desired. Further, it will be found a handy collection of tests capable of application in the preliminary investigation of many different types of problems.

There is but one direction in which the compilation might have been improved upon, and that is in respect of the character of the information given under the heading of solubility in some of the monographs. No doubt the compilers considered that from the point of view of an actual standard quantitative figures for solubility were not only unnecessary but difficult to check; nevertheless it would enhance the value of the book if precise data regarding solubility were included in some cases. This applies particularly to materials of limited solubility, such as potassium iodate or potassium hydrogen phthalate, where a statement of the actual solubility would be more helpful than the comment "slowly" or "slightly" soluble in water.

[1] "'Analar' Standards for Laboratory Chemicals: Being Improved Standards for the Analytical Reagents Formerly Known as 'A.R.'", formulated and issued jointly by The British Drug Houses, Limited, and Hopkin and Williams, Limited; 1934. Demy 8vo., pp. 295.

ANATOMY FOR DENTAL STUDENTS.

A VOLUME on anatomy for dental students has been specially compiled under the editorship of Dr. E. P. Stibbe, himself a contributor, with a view to meeting the requirements of the dental students qualifying for the licentiateship of the Royal College of Surgeons (England).[1]

As such, it is an excellent work, and as a book of reference for practitioners; but it is doubtful whether it would meet the requirements of those schools where a more extended and more detailed knowledge of anatomy is required than is the case with the Royal College of Surgeons.

The question of how great a knowledge of anatomy is required by the dental student is a very debatable one, and the argument extends also to those other foundational subjects common to students of both medicine and dentistry, such as physiology, pathology and bacteriology. In these days of wide extension of knowledge of practical and technical subjects relating to the practice of dentistry, and of congested time-tables, the tendency is to restrict the teaching of basic sciences to the minimum requirements. Whether for better or for worse, this policy seems inevitable unless an extension of the period of the curriculum is allowable. And where will this end?

For the restricted course the volume under review provides an excellently detailed description of the anatomy of the head and neck, with an outline of the central nervous system and the abdominal viscera in Part I. Part II gives a systematic and easily followed practical guide to the dissector, covering the thorax, head and neck, the whole concluding with a very full index.

[1] "Anatomy for Dental Students, Systemic and Practical", by Six Teachers, edited by E. P. Stibbe, F.R.C.S.; 1934. London: Edward Arnold. Demy 8vo., pp. 440, with illustrations. Price: 21s. net.

The Medical Journal of Australia

SATURDAY, JUNE 1, 1935.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

THE MEASUREMENT AND USE OF VITAMINS.

SINCE Gowland Hopkins first pointed out that animals cannot live on a diet of pure protein, carbohydrate and fat, with the addition of the necessary inorganic salts, our knowledge of the accessory food factors or vitamins has grown enormously. Each year something new is added to the old discoveries, and the studious medical practitioner, with all his other reading, finds it difficult to follow the course of progress. The survey of knowledge regarding vitamins, published by the Medical Research Council of Great Britain in 1932, was for this reason extremely valuable. But the knowledge of vitamins is by no means esoteric. The general public has been taught a great deal about them, and this is all to the good. People know that vitamins are essential to life and that they must be present in the daily food of the individual. They probably know that a deficiency of vitamins will eventually undermine health and may give rise to disease. Possibly they do not know that vitamins are easily destroyed and that their presence in food is in a large measure dependent on the way in which the food is prepared for consumption. Probably they do not know that the requisite amount of vitamin is infinitesimally small and that excess may give rise to hypervitaminosis with its attendant ills. It must be admitted that round the question of hypervitaminosis much argument has centred. Some workers have postulated a lethal effect from overdosage; others have found definite symptoms of toxicity; and others again have not been convinced that symptoms have been caused by the vitamin itself, and not by an associated impurity. The weight of evidence, however, definitely favours the existence of a condition of hypervitaminosis. Even if evidence pointing to hypervitaminosis did not exist, it would be reasonable to suppose that a substance capable of exerting such beneficial effects in minute quantities would, if given in excessive doses, produce harmful effects. No less an authority than Sir George Newman has opposed the indiscriminate fortifying of foods with vitamins. He is reported to have said that this practice, particularly if used in making bread and in the preparation of similar food, is likely to be a menace to health. Obviously then, if vitamins are to be added to foods, it is important to be able to determine a suitable dosage.

The effect of a vitamin-containing food was originally determined by the biological method. The rat was generally used, and laboratory workers spoke of so many "rat units" of a substance. This method was more or less satisfactory, but it became apparent before long that the results of this method were not always reliable, since different animals presented wide variations and one animal might vary considerably at different periods. The first vitamin standardization was undertaken by the Medical Research Council of Great Britain at the National Institute for Medical Research—it announced a provisional standard for vitamin *D*. Soon after this the matter was taken up by the Health Organization of the League of Nations and the First International Conference on Vitamin Standardization was held in London in 1931. Provisional standards were then set up for

vitamins *A*, B_1, *C* and *D*. The Second International Conference was held in London in 1934, and the findings of the first conference were reviewed in the light of two years' experience. Information on this subject may be gathered from the recently issued report of the Medical Research Council. At present it must suffice to state that the International Conference of 1934 did not alter the standards for vitamin B_1 and vitamin *D*. For vitamin *A* pure β carotene was adopted as the basic standard in place of the provisional standard of impure mixed carotene, and it was also decided that a subsidiary standard of cod liver oil, of which the unit value had been accurately determined in terms of the pure carotene standard, should also be prepared and held available for certain purposes. For vitamin *C* pure ascorbic acid was adopted as a standard. It will thus be seen that a higher degree of accuracy is being obtained in this most important field of investigation. The Medical Research Council is justified in its prediction that, with increasing knowledge of the chemistry and physics of vitamins, animal tests will become of less importance in evaluating the vitamin content of foodstuffs, while chemical and physical methods will become more and more readily applicable. The Council draws attention to the approval by the International Conference of a purely physical method for the estimation of vitamin *A* in liver extracts and other rich sources; this method involves the determination of the coefficient of absorption by means of a spectrophotometer and dispenses with the need for a biological test.

The facts here set out show that even now it is possible to estimate the vitamin content of foods. In time this will be more easily accomplished. What was a difficult biological investigation is becoming, and in some instances has become, a matter of chemical or physical testing. The knowledge that is available must be used in everyday practice. All the reputable houses that put concentrated vitamin products on the market are alive to the need for the application of the knowledge already gained. Many of them, indeed, have research workers engaged in trying to perfect present methods and in looking for simpler ways and means. Medical practitioners will be well advised to become familiar with the standardization of vitamins. They will then use in their practices only those preparations that come from firms of repute and that are known to conform to the standards. They will spurn the widely advertised vitamin concentrates that are supposed to cure divers chronic diseases, and will seize every opportunity of preventing self-medication that may be dangerous.

Current Comment.

ELIMINATION OF MILK-BORNE DISEASE.

MILK is frequently a vehicle for the dissemination of disease, often in epidemic form. Such diseases include typhoid and the paratyphoid fevers, tuberculosis (bovine and human), diphtheria, scarlet fever, foot and mouth disease, bacillary dysentery, septic (streptococcal) sore throat, and *Brucella* infection (undulant or Malta fever). Possibly infantile paralysis may be similarly spread. W. C. Davison states that in America, from 1881 to 1927, 791 outbreaks of milk-borne disease were reported and that many others have occurred.[1] This constitutes a truly formidable record. The United States Public Health Service has gathered information on the frequency of *Brucella* infection. In 1931, 1,545 cases were reported, and 1,407 in 1932. Other cases are recorded from England and the Continent of Europe. Probably the spread of tuberculosis by milk is less frequent than formerly. Before 1910, 25% of cattle slaughtered in London were tuberculous; 16% of samples of ordinary market milk in New York City contained tubercle bacilli. In Berlin and London the percentages were 28 and 22. In 1916, in Edinburgh, of 168 cases of tuberculosis in the young, 78·4% in children below five years of age and 70·3% in those between five and sixteen were caused by bovine bacilli. In that city also in 1912, 62% of bone and joint tuberculosis was bovine in origin. In New York in 1917, of 88 tuberculous children under five years of age, only 12·5 had bovine infections. Davison states that most cases of tuberculosis are human in origin. In England and Wales, however, it is estimated that 4,000 new cases of infection with bovine bacilli occur every year, and about 2,000 deaths are reported annually. In the transmission of bacillary dysentery contamination of milk occurs more often in the home than in the dairy. Davison details the measures adopted in the United States to reduce the incidence of milk-borne disease. He insists on simplifying the enforcement of milk sanitation and reducing its cost in order to increase milk consumption. But

[1] *American Journal of Diseases of Children*, January, 1935.

he states that the milk supply must be made absolutely safe and still be within the means of the majority of the people. He points out that in spite of the excellent results of the sanitation programme outbreaks of milk-borne disease still occur, and the expense of enforcing the provisions makes milk consumption too expensive for the majority. Again, in spite of perfect inspection of the herds and dairies, and efficient methods of pasteurization and distribution, milk may be infected in the home. Dysentery bacilli from the hands of the mother or nurse, or from flies, may induce fatal infection in the infant. In babies dying from diarrhœa the contents of the upper intestinal tract are heavily infected with colon bacilli. This is probably due to diminished pancreatic secretion owing to fever or hot weather, with accumulation of undigested food. Such food is, in Davison's opinion, infected by organisms ascending from the colon and liberating irritating products. Saprophytic colon bacilli in cow's milk may be harmful. His remedy for milk-borne disease is lactic acid milk, which is bactericidal. Buttermilk as an infant's food was advised by N. Rosen von Rosenstein in 1776, but soon fell into oblivion. It was not reintroduced into Holland till 1865. It was introduced into Germany in 1902, France in 1907, and the United States of America not until 1919. Many explanations have been advanced for the beneficial results of lactic acid milk. K. B. Rothey showed that dysentery and typhoid bacilli introduced into lactic acid milk were soon destroyed. B. U. Brooks reported that the death rate of coloured infants in one city fell from 196 to 119 per thousand births, and of white infants from 83 to 57 during four years by lactic acid milk. Davison considers that whole lactic acid evaporated milk, containing 7·5% of added carbohydrate and 0·75% of lactic acid, is the best artificial infant's food. Cane sugar is a satisfactory carbohydrate, but molasses may be used instead, the latter having the advantage of containing iron and calcium, but sometimes causing distension, loose stools or vomiting, especially in weak or premature infants. If more than 7·5% of carbohydrate be added, the infant's stools may be loose; if less than 7·5%, constipation may result. The mixture contains one calorie per cubic centimetre. The addition of carbohydrate is unnecessary for infants over eight months of age. Davison states that infants' foods should be judged by five criteria, all of which are satisfied by whole lactic acid evaporated milk. Infants thrive on such milk as well as on breast milk. Vomiting, diarrhœa and bacillary dysentery are less in infants so fed than on any other food. The evaporation process employed removes the risk of infection by organisms which may be spread from a dairy. Safety in home handling and in the infant's intestines is assured. No other infant food possesses the same bactericidal power. The antibacterial or lysozyme strength of breast milk is feeble in comparison. Sweet milk mixtures, even if boiled in the nursing bottle, may be infected by the mother's hands while the nipple is being attached. Whole lactic acid evaporated milk cannot be readily infected in the home or in the upper intestinal tract. Finally, such milk is universally obtainable and is cheap. Vitamin loss can be overcome by tomato or orange juice or cod liver oil.

Davison is an earnest advocate of this preparation, but whether it would be practicable in the remote country and inland districts of Australia is by no means certain. Nor is it certain that the continued use of lactic acid would be altogether harmless. In dyspepsia due to carbohydrate fermentation lactic acid is useless; it may aggravate the condition. In olden times, when given to diabetics, lactic acid often produced severe "rheumatic" symptoms. It often causes epigastric pain and flatulence. L. G. Parsons does not advocate the use of lactic acid in children under three months of age. J. A. Stephen and E. R. C. Walker consider it unsuitable in cases of acidosis and anhydræmia. The vogue of lactic acid, as produced by the various lactic acid bacilli, comes in cycles with partial or complete oblivion in the intervals. A small practical point is that while the preparation of lactic acid milk is theoretically easy, in practice a tendency to curdle is often overcome only with difficulty.

PELLAGRA.

THE view held by the majority is that pellagra is a deficiency disease, caused by a lack of vitamin B_2. It occurs most frequently in maize-eating communities, but may occur anywhere. Recently some doubt has been cast on the vitamin theory. Interest in the disease is enhanced by the reports of so-called secondary pellagra. This interesting disease is discussed in a recent paper by S. Levy Simpson.[1] He defines it as "pellagra occurring secondary to an initial gastro-intestinal lesion or disorder, the causation of which is entirely unconnected with pellagra"; he states that it is comparable with the megalocytic hyperchromic anæmias resulting from gastrectomy and other lesions of the alimentary tract. He describes a case in which partial gastrectomy had been performed for gastric ulceration; the patient developed typical symptoms and signs of pellagra. The addition of vitamin B_2 only to the diet caused rapid and great improvement in the skin lesions, and, after fifteen days, when the patient was given a full diet, rich in vitamin B_2, improvement continued. Simpson suggests that if pellagra is regarded as a deficiency disease, the existence of intrinsic and extrinsic factors may be postulated. A number of ways for the causation of secondary pellagra may then readily be conceived. He suggests that minor manifestations of the disease might be frequently missed, and advises the use of yeast or "Marmite" in prophylaxis. The most interesting feature of the paper is the theory of causation. We agree that pellagra, whether "secondary" or not, and even if it is well established, might readily be overlooked. Medical practitioners in Australia should bear it in mind; it may not be so rare as it is usually supposed to be.

[1] *The Quarterly Journal of Medicine*, April, 1935.

Abstracts from Current Medical Literature.

PÆDIATRICS.

Pollen Sensitiveness in Asthmatic Children.

R. BRUCE PEARSON (*Archives of Disease in Childhood*, December, 1934) investigated 250 children attending the asthma clinic at Great Ormond Street Hospital with a view to finding the frequency with which pollen sensitization occurred in asthmatic children. He points out that, although pollen is well recognized as a common cause of symptoms of allergy, the frequency with which the sensitiveness to it is manifested by hay fever tends to detract from its importance as a causative factor in asthma. The tests indicate that pollen sensitization is of more importance than is generally recognized. In 42%, positive intradermal reactions to grass pollen extracts were obtained. This figure compares with 60% for a series of the same age group tested with chicken feather extract in comparable dilution. Information of value in the treatment of asthma is thus afforded. Avoidance of pollen may be just as helpful to asthmatics as the routine removal of feathers and hair-containing materials from their environment. A move to the country might be responsible for an increase in the number of asthmatic attacks in pollen-sensitive children, who are not uncommonly free from their symptoms at the seaside during the pollen season. Finally, the treatment of pollen-sensitive asthmatics by injections of pollen extract is likely to be followed by a decrease in the frequency and severity of attacks.

Electrocardiography in Chronic Rheumatic Heart Disease.

ELIZABETH BRAKELY (*Archives of Pediatrics*, December, 1934) studied the electrocardiograms of 100 consecutive children attending a heart clinic, to which a number of the children had been referred on account of heart murmurs discovered during the course of routine examinations by school physicians or in health clinics. The object of the investigation was to ascertain if the electrocardiography would give any information not furnished by physical examination concerning children with milder forms of rheumatic heart disease or with heart murmurs which may or may not mean organic heart changes, as compared with patients with definite rheumatic heart disease. The author concludes that in these children the electrocardiogram differs little from that found in normal children. In doubtful cases of mitral stenosis it may be of assistance in diagnosis. The most important changes to look for in rheumatic heart disease are increase in height and breadth of the *P* wave, accompanied by notching, increase in length of the *P-R* interval, slurring or notching of the *Q R S* complex, inversion of the *T* wave in two or more leads, and right axis deviation.

Types of Pneumococci in Infections other than Pneumonia.

R. W. FAIRBROTHER (*Archives of Disease in Childhood*, February, 1935) points out that the pneumococcus is not infrequently responsible for infections other than pneumonia, and that the typing of the causative organism is consequently of some importance. Empyema is more commonly caused by pneumococcus of Type I than by any other type. In the author's series 75 patients were examined; these included 59 with empyema, 12 with meningitis, and 4 with other conditions. Of the patients with empyema, 87% were infected with pneumococcus of Type I. None of the patients had received any serum in the treatment of the preceding pneumonia. As it is now generally accepted that pneumonia of Type I responds well to serum therapy, it is suggested that as early serum therapy of this type of pneumonia becomes more widely applied, the incidence of empyema should decrease.

Iron Therapy During Infancy.

HOWARD L. ELDER (*Archives of Pediatrics*, November, 1934) discusses the advisability of giving iron as a routine measure during infancy. He refers to the many articles published each year on the frequency of anæmia during infancy and the methods used for treating the condition; but he draws attention to the apparent absence of any method in general use for its prevention. Those familiar with modern medicine realize the value of preventing rather than of curing disease; frequently the method employed in treatment is also useful in prevention. The author refers to the fact that patients with scurvy were relieved or cured when given fresh fruit and vegetables; but it was only in comparatively recent years that this procedure was adopted as a routine during infancy, and as a result scurvy has become almost extinct. Similarly, it was found that children with rickets definitely improved by correcting the diet and giving cod liver oil, and some form of vitamin *D*. The necessity of including vitamin *D* in the infant's dietary *régime* is now well recognized. During recent years much work has been carried out on calcium metabolism, and it has been shown that large amounts of this mineral are available as a result of its storage in the osseous tissues. This serves as a sufficient reserve, which is available to meet the demands of the other tissues. As nutritional anæmia occurs frequently during the first year of life and is treated by the administration of iron, and in view of what is known regarding the storage of other minerals and vitamins, the author asks whether it is not advisable to adopt the routine prescription of iron for the infant to prevent anæmia. His observations over a number of years have led him to four conclusions in this respect, namely: that iron should always be prescribed during infancy as a means of preventing anæmia; that present methods of diagnosing anæmia fail to make allowance for the absence of available iron in reserve; that it is necessary to supply iron beyond the amount required by the circulating blood so that a reserve will be built up in the tissues; and that the treatment of anæmia should be carried on long after a normal hæmoglobin value of the blood has been attained. The author's formula for the administration of iron in infancy is one pint of cod liver oil emulsion added to thirty grammes of saccharated ferrous carbonate. One teaspoonful of this is given twice a day from the age of ten weeks till the age of six months; thereafter a teaspoonful a day is continued indefinitely.

Papular Urticaria.

B. C. WHITE (*Archives of Disease in Childhood*, February, 1935), in an analysis of 299 patients from the Children's Hospital, Birmingham, has undertaken an inquiry into the ætiology of *lichen urticatus*. He refers in the first place to the fact that, although the condition has been accepted almost universally in recent years as a manifestation of allergy, there is still considerable difference of opinion as to the nature of the allergen. Foods, particularly proteins, but also carbohydrates and fats, (Bray) have been incriminated. On the other hand, Hallam is of opinion that the cause of *lichen urticatus* is not food, but some environmental factor. In regard to the nature of the condition, the author points out that, although it bears resemblance to both the ordinary urticaria of adults and Hebra's prurigo, papular urticaria is a distinct morbid process and separable from both these conditions. An urticarial rash does appear during the evolution of its lesions; but the papule is an additional lesion, which cannot be evoked by the urticaria-producing agent, *H*-substance, and is different from the papule in Hebra's prurigo. Although not especially confined to any particular age period, papular urticaria is essentially a disease of the first four years, beginning most frequently during the first nine months of life. The occurrence of other allergic manifestations, either in the personal or family history, is no more frequent than in a series of normal people. Dentition is probably a definite predisposing factor. Digestive disorders may also predispose to the condition; but their importance has been over-estimated. Papular urticaria is aggravated by heat and a diet containing too much carbohydrate; but there is no evidence that it is a manifestation of food allergy. In regard to environmental influences, there are two outstanding

facts. In the first place it is remarkable that among patients in children's wards under treatment for different digestive disorders and receiving a variety of diets papular urticaria is an event of the greatest rarity. Secondly, the admission of patients to hospital always brings relief, and even a change of residence often has a striking effect. It would therefore appear that the exciting agent is something connected with the patient's home environment; but its precise nature remains obscure. In treatment it is advisable to pay attention to clothing and ventilation, in view of the influence of heat on the symptoms; to restrict the carbohydrate content of the diet; and to prescribe rhubarb and soda. Admission to hospital is by far the most valuable therapeutic measure in all severe and persistent cases.

ORTHOPÆDIC SURGERY.

Fracture of the Phalanges.

F. L. SMITH and D. L. RIDER (*The Journal of Bone and Joint Surgery*, January, 1935), in a study of the healing of phalangeal fractures, state that all compound fractures were given immediate surgical attention, the major portion of which was directed to the repair of the skin and soft tissue. All cases in which there was devitalization of soft tissue or gross contamination with dirt, careful *débridement* was carried out, and when the wounds were relatively clean they were treated with suitable antiseptics and immediately converted into closed fractures. If the nail was badly lacerated or partly torn, it was removed. The authors consider it always advisable to preserve the nail whenever possible, as it acts as a natural splint. Tongue depressor splints were employed in about half the number of patients. Although often they were not necessary for fractures in the distal phalanges, they were used for protection. Splints were kept on for about two weeks; in a few cases it was necessary to keep the splints longer. In those cases in which, from the reading of the skiagram, the small fragment of bone appeared to be insignificant and which were accompanied by an inability to extend the distal phalanx and, if allowed to go on, would have resulted in a permanent drop joint, they found the use of a small splint with the distal phalanx in hyperextension for at least thirty days gave better results. As shown by the author's table, the time for clinical healing is much less than the time required for the appearance of solid bony union in a skiagram. The time for clinical healing was arrived at by computing the number of weeks which elapsed from the time of injury to the time when the patient was able to return to his former occupation and was therefore dismissed from further clinical observation. It was found that fractures of the proximal phalanges required a somewhat shorter time for healing as shown by radiographic examination, although on an average there was little difference in time required for the healing of fractures in the distal, middle and proximal phalanges, the average being about five months.

End-Result Study of Humeral Shaft Fractures.

H. ROGERS (*Surgery, Gynecology and Obstetrics*, December, 1934) thinks that the end-results of fractures of the humerus intelligently treated by any suitable method are excellent. Generally transverse fractures should be reduced by manipulation (closed or open) and held by fixation (external or internal); oblique fractures should be reduced and held by constant traction; comminuted fractures should be treated by the simplest method suited to the individual. He is of the opinion that constant traction is dangerous in transverse or short oblique fractures, especially where they occur in the middle and lower thirds, and should be used only with great caution, if at all. He considers that, when necessary, bed traction is more effective than ambulatory traction, that the former may be applied by means of the Thomas arm splint or Blake's suspension, and the latter by Jones's humerus splint. The indications for early operative reduction are failure to accomplish or maintain satisfactory reduction by closed methods and anticipated non-union. Bone plating alone will not bring about secondary union after the original reaction of repair has subsided, as it will in a fresh fracture. Bone grafting is usually necessary. Persistent delayed union and non-union are the greatest obstacles to the successful operative treatment of old ununited fractures. The patient should be given the maximum chance for union at the time the first operation is performed by the use of a radical procedure combined with the vigorous employment of the various common systemic aids. In a transverse or short oblique fracture in the middle or lower third of the humeral shaft in which there has been no contact between the bone ends during the early period of active reaction of repair, non-union may reasonably be anticipated. Radial nerve injury rarely results in permanent disability.

Chronic Generalized Fibromyositis.

A. M. ORNSTEEN (*Annals of Surgery*, December, 1934) describes some cases of gradually increasing rigidity and difficulty in walking, resulting from fibromyositis. Rigid spine and the myotonic stage were characteristic features, the myotony improving after several moments of walking. The author describes Batten's classification of the inflammatory diseases of muscles as follows. (*a*) Primary infections: (i) polymyositis, including acute polymyositis, dermatomyositis, hæmorrhagic myositis, polymyositis with *erythema multiforme* and urticaria and pseudotrichinosis; (ii) neuromyositis; (iii) tuberculous myositis; (iv) syphilitic myositis; (v) myositis due to *Trichinella spiralis*. (*b*) Secondary infection in the course of acute or chronic disease: (i) myositis in the course of specific fevers, as typhoid, typhus, smallpox; (ii) infective myositis occurring in pyæmia, infective endocarditis, glanders, gonorrhœa, puerperal infection and infected wounds, actinomycosis, erysipelas *et cetera*. (*c*) Myositis with special terminal lesions: (i) *myositis ossificans progressiva*; (ii) *myositis fibrosa*, general or local. He discusses the differential diagnosis under these headings. He is unable to find authentic reports of the ætiology of *myositis fibrosa*. He quotes Pemberton as stating that most authors include fibrositis, myalgia and even myositis under the term "myositides" and that he (Pemberton) is of the opinion that the pathological process implied is really part of and belongs to the rheumatoid or arthritic syndrome, and that there is every reason to believe that it springs for the most part from the factors causative also of arthritis, and should be regarded as an expression in the muscular tissues of the same underlying process.

Experimental Bone Regeneration.

W. J. STEWART (*Surgery, Gynecology and Obstetrics*, December, 1934), in attempting to perfect a means of stimulating bone growth by using lime salts and autogenous grafts as sources of available calcium, found that boiled bone grafts died and that when they were inserted into defects of the radius there was no production of new bone about or within them; he also found that the only source of new bone formation in these experiments was from the ends of the radius. Small fragmented live grafts similarly used were the sources of large amounts of new bone growth in each instance. Throughout the entire series of experiments, wherever a small bone chip was left behind at operation there was evidence of active bone growth from it. There was failure of regeneration of the shaft after lime salts had been implanted in the radial defects. The effect of mixing traumatized muscle and lime salts in the radial defects was to create a few areas of calcification in necrotic tissue. No regeneration of the shaft took place, and the only constant effect of implanting lime salts in carpal and tarsal defects was the production of a definite proliferative arthritis. No bone was laid down in the defects. The author concludes that lime salts and boiled bone, when placed into a bone defect with either traumatized muscle or fascia, do not serve as a source of available calcium, resulting in supersaturation of connective tissue and regeneration of missing bone; and that live bone chips placed in bone defects regenerate the missing bone.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held in the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, on April 24, 1935, DR. A. M. DAVIDSON, the President, in the chair.

Immunization against Diphtheria and Scarlet Fever.

DR. HILDA BULL, of Melbourne, read a paper entitled "Immunization against Diphtheria and Scarlet Fever" (see page 671).

DR. A. M. DAVIDSON complimented Dr. Bull on the comprehensiveness of her paper and on the extent of the research that it implied. He declared the paper open for discussion by members.

DR. PHYLLIS ANDERSON said that *Corynebacterium diphtheriæ* was an organism of saprophytic habits, elaborating, as far as was known, one diffusible exotoxin. The nature of the toxin was readily studied, as there were susceptible laboratory animals at hand. The existence of active immunity could be measured by means of the Schick test, in terms of circulating antitoxin against a known toxin, and long experience had shown the level which insured immunity against the average amount of infecting droplet contact amongst the general population. Immunity was, however, a relative term, and might break down in the presence of massive infection.

Natural immunity was dependent on the carrier state and subclinical infection in those previously susceptible. There were two methods, therefore, of attempting control of the disease: first, bacteriological, by the discovery and elimination of carriers, which would abolish the risk of massive infection but produce ultimately a susceptible population; and, secondly, active immunization of the infant population to the toxins of the bacillus. The means and practicability of bringing this about had been dealt with by Dr. Bull.

The Health Committee of the League of Nations had formally adopted the principle of active immunization "not later than the pre-school period after the end of the first year of life".

"Charitable organizations and administrations receiving children in homes are advised to require from both children and staff a diphtheria immunization certificate on admission, or a certificate attesting that the Schick reaction is negative."

"In the opinion of experts, diphtheria immunization should form the subject of active public education on the part of health administrations of the different countries in order to bring home to everyone the advantages of this method of protecting the public health."

The hæmolytic streptococcus, on the other hand, was an organism of much greater complexity; it infected locally and produced an erythrogenic toxin which became neutralized; later it might invade the blood stream and pyogenic effects appeared, or it might produce a further toxin which selected the renal glomeruli for its region of activity.

The Dick test was a visible demonstration of the individual's response to one evidence of hæmolytic streptococcus infection, namely, the rash-producing toxin. The power to neutralize it might reside in persons who were still susceptible to one or all of its other phases of activity. Kinloch, of Aberdeen, in recording the results of immunization of nurses, said that though clinical scarlet fever disappeared from the staff, the incidence of streptococcal tonsillitis actually increased.

The use of antitoxin in the disease itself could neutralize the circulating toxin, cause defervescence, and produce a marked improvement in the condition of the patient. But they knew from experience that the later manifestations of the disease could not thus be cured. Indeed, how could they expect, when in untreated cases in which the patient produced his own Dick negativity and renal and septic complications still appeared, that the conferring of passive immunity for the first week, which was proven frequently to relapse to the Dick-positive state, should abolish the likelihood of these complications?

The production of active immunity to the toxins of the hæmolytic streptococcus had not yet been accomplished. The production of Dick negativity meant only one thing, namely, that the person infected would not exhibit a rash—the warning flag would not fly.

The pædiatrician knew only too well the story of the child who had an inconspicuous sore throat, which had not occasioned alarm, let alone limitation in its activity or diet, who was suddenly found to be pale, œdematous, and passing small amounts of urine loaded with blood.

The *Corynebacterium diphtheriæ* was an enemy who fought with a single weapon, whereas the hæmolytic streptococcus was a very Napoleon amongst microbes—you might defeat him at Austerlitz and exile him to Elba, but he would come back with new weapons, and that last battleground, like the field of Waterloo, would be scarred to the end of time.

PROFESSOR H. K. WARD said that they should be grateful to Dr. Bull for the pioneering work that she had done in the prevention of diphtheria; he understood that such work was wanted badly. The scientific facts concerning diphtheria were well known, but between the knowledge and its application there was always a lag. And when it was a question of public health other factors came in, such as the difficulty of obtaining the confidence and support of the general public. If the disease was a dramatic one of which people were afraid, the lag period would be short. If many persons were to develop virulent diphtheria, there would be large numbers wishing for protection. But when the disease was mild, opportunities for general immunization depended on the education of the community. It was largely a question of the education both of the medical profession and of the public; and it was also a question of economy. Professor Ward asked whether Dr. Bull could give figures to indicate the amount of money spent *per capita* on public health in her community as compared with other communities. It would be interesting to see whether the State was doing its share in helping to supply adequate health measures. Ultimately this question was one of pounds, shillings and pence; and a question of whether the community could be persuaded to pay for protection against disease.

In regard to the hæmolytic streptococcus, Professor Ward agreed with all that Dr. Anderson had said.

Dr. Bull had asked about the effect of the Dick test in expectant mothers. The test had been carried out, but not on large numbers of patients. And Professor Ward thought that the evidence obtained did not show conclusively that there was a lower incidence of puerperal sepsis among the mothers who gave a "negative" reaction.

DR. H. A. RIDLER, in thanking Dr. Bull for her paper, said that much that she had said was beyond him. He was interested in scarlet fever and puerperal sepsis. In regard to immunization, he would restrict his remarks to the form of sepsis in which the condition was like that obtaining in scarlet fever, but was regarded as puerperal sepsis. Dr. Ridler mentioned a case of this type that Dr. Gibson had seen. The patient died, Dr. Gibson became ill, the attending maternity nurse became ill, and he himself, though he saw the nurse only once and put her into hospital, became ill. Dr. Ridler asked Dr. Bull whether persons immunized against scarlet fever had any immunity against puerperal sepsis.

Lately much money had been collected for the reduction of maternal mortality. How was this to be spent? The question of the hæmolytic streptococcus and scarlet fever might have some money spent on it; for puerperal sepsis accounted for the largest percentage of deaths during maternity.

DR. A. H. TEBBUTT said that he had enjoyed Dr. Bull's address and was sorry to say that he agreed with nearly all of it. He liked to hear a paper which worked him up to express his disapproval; he thought that Dr. Bull had said all that there was to be said. In regard to

immunization against scarlet fever, Dr. Tebbutt had, like Dr. Bull, found that he could not give large doses of Dick toxin (Fairfield strain). He found that if he started with 125 skin test doses a moderately severe reaction occurred in a few boys, and he could not go higher for the initial dose. He followed the initial dose by 250 and then 500 skin test doses. Having agreed to immunize these boys with three doses, Dr. Tebbutt did not feel justified in going on. This was a difficult problem, because big doses could not be given at first, and the number of injections necessary militated against the complete carrying out of the immunization. One boy who was given the three doses described developed mild scarlet fever within three months. Dr. Tebbutt agreed that in deciding on the question of immunization one had to regard slight reactions as positive reactions, and immunize the children who gave them.

In regard to the case of sore throat due to a hæmolytic streptococcus, but without rash, following immunization, quoted by Dr. Bull, Dr. Tebbutt felt doubtful of looking on this as a failure. It was known that immunization with Dick toxin did not produce immunity to sore throat. The occurrence of sore throats after immunization was not less frequent in some centres where this had been investigated. He would regard only those cases in which scarlet fever occurred as indicative of failure.

Referring to the questions with which Dr. Bull had concluded her paper, Dr. Tebbutt said that they were all problems that no one could answer. He had no doubt that they were justified in giving large doses of antitoxin in streptococcal infections other than scarlet fever. He had seen good results follow the combined administration of streptococcal antitoxins and the modern polyvalent anti-streptococcal sera in large doses in infections due to hæmolytic streptococci, including puerperal streptococcal infection, and even in cases in which the clinical prognosis was extremely bad. Large doses were advised, and he preferred the intravenous route. He felt that they were justified in persevering with treatment of this kind until other more satisfactory methods were available.

DR. H. G. WALLACE congratulated Dr. Bull on her paper, and particularly on having a council willing to enable her to carry out work of this sort; for in the last resort the question boiled down to one of pounds, shillings and pence. In New South Wales there had been over 40,000 cases of diphtheria in ten years and over 1,000 deaths. Last year, in a big epidemic, the cost was £50,000 in treating 6,000 odd patients, which worked out at about £8 per patient. If sufficient interest could be created in this scourge to spend one-tenth of this sum on prevention, it would be possible to reduce the incidence of the disease and to produce results like those obtained in Melbourne. New South Wales had been lagging behind the other States. Victoria had been up against the same problems: the apathy of country councils and of local health authorities. Queensland was more fortunate; the council in Queensland was responsible for the treatment of infectious disease, and in these circumstances the council found that it paid to prevent diphtheria. In New South Wales the Hospitals Commission was in most cases responsible and there was no cost to the councils. In Queensland, in order to induce the councils to take the matter over, the Government, for a short period, offered them free immunization; this gave a big fillip to the work. Dr. Cilento had said recently that about 80,000 children had been immunized in Queensland. In New South Wales the numbers were small; immunization was commenced in about 1924, as it was in Melbourne; but after the Bundaberg tragedy it had been dropped. Now it had been started again, but there was only a meagre response from the public. For the practical application of these measures it was necessary to enlist the services of the general practitioner. Moreover, such measures were a great expense to any health department dealing with large numbers. Dr. Wallace hoped that the services of general practitioners would be enlisted, and that they would realize that immunization was harmless, safe and effective. If this could be done, then people might be induced to bring their children along as those born reached a susceptible age. Unless something of this sort were done no progress would be made in stamping out diphtheria. It was necessary to secure the assistance of the British Medical Association and of the general body of general practitioners. It was possible in an institution to wipe out diphtheria; but to do much for the community as a whole a big effort was necessary. People did not know much about anatoxin and were rather afraid of it; moreover, some practitioners had even been known to advise their patients against it. No case of death had ever been found to be due to anatoxin. Dr. Wallace was interested in allergic reactions to the antigen. In Queensland Dr. St. Vincent Welch told him that he had never seen a case in which a dangerous reaction had occurred. Dr. Wallace would like Dr. Bull to give figures for such reactions in Melbourne. In New South Wales there had been a few sore arms in about 1% or 2% of cases. Dr. Wallace asked Dr. Bull what she considered to be a positive Moloney test, what was the reaction, and what was the size of the dose of anatoxin given to reactors. It seemed that Melbourne had set a very good example, and Dr. Wallace wished that means might be suggested whereby interest might be awakened in New South Wales.

Dr. Bull, in reply first to Professor Ward, said that she could not tell him what was the *per capita* expenditure on public health in Melbourne. In a community of 90,000 the cost of the actual immunization was very little; far less than if it were carried out for individuals by private practitioners. Moreover, the salaries of public health officers were not princely. Dr. Bull said that it cost very little to immunize a number of children, perhaps about one shilling per head. A Schick test would cost only one-fiftieth of half a crown. If a full-time officer were doing the work, it was cheaper. Nevertheless, Dr. Bull agreed that the general practitioner in his capacity as family doctor, and also in all outlying places, should be prepared to immunize the children under his care.

Dr. Bull said that it was reported that in Toronto the manufacture of antitoxin had practically stopped, and that in Montreal over 50% of the child population had been immunized. Some years ago diphtheria was a very serious disease in Canada, but its incidence had greatly diminished. Dr. Bull said that it was very difficult to educate the public. It was all very well to say that if the children would not come and be tested they could act as controls. Dr. Bull thought that it would be better if the children did not have fathers. It was always the fathers who objected to immunization, apparently as a show of authority.

Dr. Bull said that Dr. Killick Millard, in speaking recently of immunization, considered that if the number of immunes were increased the number of carriers would also be increased, with added danger to others. Some people said that if it were not possible to get the requisite number immunized, why do it at all? But Dr. Bull thought that in any case it was worth while, both as an educational measure and as a protection to the individual.

When the Moloney test was performed and the reaction extended more than half an inch it was to be regarded as positive. If the extent was less than the standard size, a dose of 0·1 or 0·2 cubic centimetre should be given, according to the severity of the reaction. The most satisfactory work was carried out at the infant welfare centres.

DR. A. M. DAVIDSON said that it had been an instructive evening. He himself, with his experience in general practice, realized the trouble that they were up against in the public indifference and the fear of having anything injected under the skin. Speaking on behalf of the Council of the New South Wales Branch of the British Medical Association, Dr. Davidson thought that they had made a happy start for the scientific meetings of 1935 in having someone of Dr. Bull's standing from another State to address them. Her paper had been most helpful and he wished to express to her his gratitude for the trouble that she had taken in preparing it, and also to the Public Health Department in Melbourne his appreciation for having made Dr. Bull available to lecture to them.

ANNUAL MEETING, MELBOURNE, 1935.

THE annual meeting of the British Medical Association will commence at 9 a.m. on Monday, September 9, 1935, and will continue during the following four days. Registration will commence at the University of Melbourne at 9 a.m. on September 9; the ladies' reception rooms at the Oriental Hotel, Collins Street, Melbourne, will be opened at the same time. The annual dinner of the Association will take place at 7.15 p.m. on Thursday, September 12. The popular lecture will be delivered by the Right Honourable Lord Horder, on September 13 at 8 p.m. An official religious service will be held at Saint Paul's Cathedral, Melbourne, on Tuesday, September 10, at 4.20 p.m.

Numerous excursions have been arranged for visitors.

There will be fourteen sections, which will meet on Wednesday, September 11, Thursday, September 12, and Friday, September 13.

Dr. J. P. Major, the Local General Secretary, reports that there will be at least 250 visitors from Great Britain, Canada, South Africa and India.

OFFICE-BEARERS AND PROGRAMMES OF SECTIONS.

The Sections of Medicine, Surgery, Obstetrics and Gynæcology, and Radiology and Radio-Therapeutics will meet on three days; the Sections of Diseases of Children, Neurology and Psychological Medicine, and Public Medicine will meet on Wednesday, September 11, and Friday, September 13; the Sections of Ophthalmology, Orthopædics, Medical Sociology, and Pharmacology, Therapeutics and Anæsthesia will meet on Thursday, September 12, and Friday, September 13; the Sections of Pathology and Bacteriology, and Oto-Rhino-Laryngology will meet on Wednesday, September 11, and Thursday, September 12; the Section of Dermatology will meet on Wednesday, September 11.

SECTION OF MEDICINE.

Office-Bearers.

President: The Right Honourable Lord Horder, K.C.V.O., D.C.L., M.D., F.R.C.P. (London).

Vice-Presidents: Dr. J. Crighton Bramwell, M.D., F.R.C.P. (Manchester), Dr. A. W. Holmes à Court, M.D., F.R.C.P. (Sydney), Professor C. G. Lambie, M.C., M.D., F.R.C.P. (Sydney), Sir James Purves Stewart, K.C.M.G., C.B., M.D., F.R.C.P. (London).

Honorary Secretaries: Dr. S. O. Cowen, M.D. (12, Collins Street, Melbourne, C.1), Dr. J. G. E. Hayden, M.D., M.R.C.P. (55, Collins Street, Melbourne, C.1), Dr. J. C. Matthews, M.C., M.D., F.R.C.P. (Hazelacre, Downton, Wilts).

Programme.

Wednesday, September 11.

10 a.m.—Discussion on obesity (ætiology and metabolism), to be opened by Professor C. G. Lambie (Sydney). "The Treatment of Obesity", by Dr. J. H. Anderson (Ruthin Castle).

12 noon.—"The Significance of 'Gallop' and Other Types of Triple Rhythm", by Dr. Crighton Bramwell (Manchester).

Thursday, September 12.

10 a.m.—Discussion on the differential diagnosis and treatment of severe anæmia, to be opened by Dr. J. C. Matthews (Downton). "The Treatment of Pernicious Anæmia", by Dr. J. H. Anderson (Ruthin Castle). Dr. C. T. C. de Crespigny (Adelaide).

12 noon.—Occasional papers.

Friday, September 13.

Combined meeting with Section of Surgery. Discussion on thyreotoxicosis, to be opened by Lord Horder (London) and Sir Thomas Dunhill (London), followed by Professor Hercus (New Zealand), Dr. Hume Turnbull (Melbourne), Dr. A. W. Holmes à Court (Sydney), Sir Carrick Robertson (Auckland, N.Z.), Dr. Alan Newton (Melbourne).

SECTION OF SURGERY (INCLUDING UROLOGY).

Office-Bearers.

President: Sir Thomas Dunhill, K.C.V.O., C.M.G., M.D., F.R.A.C.S. (London).

Vice-Presidents: Dr. Clifford Morson, O.B.E., F.R.C.S. (London), Sir Henry Newland, C.B.E., D.S.O., M.S., F.R.A.C.S. (Adelaide), Dr. F. C. Pybus, M.S., F.R.C.S. (Newcastle-on-Tyne), Sir Carrick Robertson, M.B., F.R.C.S. (Auckland, N.Z.).

Honorary Secretaries: Dr. A. E. Coates, M.D., M.S. (3, Linda Crescent, Hawthorn, E.2), Dr. H. C. Trumble, M.C., M.D., Ch.B., F.R.C.S. (19, Collins Street, Melbourne), Dr. Lambert C. Rogers, F.R.C.S., F.R.A.C.S., F.A.C.S. (British Post-Graduate Medical School, Hammersmith Hospital, Ducane Road, London, W.12).

Programme.

Wednesday, September 11.

10 a.m.—Discussion on surgery of the pancreas, to be opened by Dr. S. Finch (England), to be followed by Sir H. Newland (Adelaide), Dr. Balcombe Quick (Melbourne).

11.15 a.m.—Discussion on hydatid disease, to be opened by Professor H. Dew (Sydney), followed by Dr. B. Kilvington (Melbourne), Dr. K. Fairley (Melbourne), Dr. K. Stuart Cross (Melbourne).

Thursday, September 12.

10 a.m.—Discussion on prostatectomy, to be opened by Dr. Clifford Morson or Dr. A. H. Burgess (England), followed by Dr. John Tait (Melbourne), Dr. Frank Macky (Auckland, N.Z.), Dr. G. H. Burnell (Adelaide).

11.30 a.m.—Discussion on carcinoma of the colon, to be opened by Sir Chas. Gordon Watson (England), followed by Dr. F. C. Pybus (Newcastle-on-Tyne), Dr. H. B. Devine (Melbourne), Dr. H. S. Souttar (London), Dr. Victor Hurley (Melbourne).

Friday, September 13.

(Combined with Section of Medicine.)

10 a.m.—Discussion on toxic goitre, to be opened by Lord Horder (London) and Sir Thomas Dunhill, followed by Professor Hercus (New Zealand), Dr. A. W. Holmes à Court (Sydney), Sir Carrick Robertson (New Zealand), Dr. Hume Turnbull (Melbourne), Dr. Alan Newton (Melbourne).

SECTION OF OBSTETRICS AND GYNÆCOLOGY.

Office-Bearers.

President: Dr. J. S. Fairbairn, F.R.C.S., F.R.C.P., F.C.O.G., (London).

Vice-Presidents: Professor R. Marshall Allan, M.C., M.D., F.R.C.S., F.R.A.C.S., F.C.O.G., F.A.C.S. (Melbourne), Dr. J. Bright Banister, M.D., F.R.C.S., F.R.C.P. (London), Dr. Ninian McI. Falkiner, M.D., F.R.C.P.I., F.C.O.G. (Dublin), Professor J. C. Windeyer, M.D., Ch.M., F.R.A.C.S., F.C.O.G. (Sydney).

Honorary Secretaries: Dr. Robert Fowler, O.B.E., V.D., M.D., F.R.C.S. (85, Spring Street, Melbourne, C.1), Dr. A. Roberta Donaldson, M.B., Ch.B. (89, Collins Street, Melbourne, C.1). (English secretary to be appointed.)

Programme.

Wednesday, September 11.

10 a.m.—Discussion on the present position of Cæsarean section in obstetric practice, to be opened by Dr. J. Bright Banister (London), followed by Professor J. B. Dawson (Dunedin, N.Z.), Dr. W. Ivon Hayes (Melbourne), Dr. H. A. Ridler (Sydney). Discussion on *placenta prævia*, to be opened by Sir Comyns Berkeley (London), followed by Professor J. C. Windeyer (Sydney), Dr. A. M. Wilson (Melbourne).

Thursday, September 12.

10 a.m.—Discussion on the prevention and prognosis of the late toxæmias of pregnancy, to be opened by Dr. J. S. Fairbairn (London), followed by Dr. John S. Green (Melbourne), Dr. F. Brown Craig (Sydney). Discussion on the remote results of puerperal sepsis, to be opened by

Sir Ewen Maclean, followed by Dr. Rupert Magarey (Adelaide), Dr. Rupert Furber (Sydney).

Friday, September 13.

10 a.m.—Discussion on the ovarian cycle and its relationship to endocrinology, to be opened by Dr. Ninian McI. Falkiner, followed by Dr. Bruce Mayes (Brisbane), Dr. F. A. Maguire (Sydney), Dr. R. F. Matters (Adelaide). Discussion on some aspects of heart disease complicating pregnancy, to be opened by Dr. H. C. E. Donovan, followed by British representatives.

SECTION OF RADIOLOGY AND RADIO-THERAPEUTICS.

Office-Bearers.

President: Not yet appointed.

Vice-Presidents: Dr. L. J. Clendinnen, M.B., B.S. (Melbourne), Dr. R. A. Gardner, M.B., D.M.R.E. (Cairo), Major D. B. McGrigor, O.B.E., M.B., D.M.R.E. (Frinton-on-Sea), Dr. H. M. Moran, M.B., F.R.C.S., F.R.A.C.S. (Sydney).

Honorary Secretaries: Dr. F. G. Stephens, M.B., B.S. (12, Collins Street, Melbourne), Dr. A. J. G. Mackay, M.B., F.R.C.S., D.M.R.E. (Radiological Clinic, Parliament Place, East Melbourne, C.2), Dr. B W. Windeyer, M.B., F.R.C.S. (The Middlesex Hospital, London, W.1).

Programme.

Wednesday, September 11.

10 a.m.—Discussion on radiation treatment of carcinoma of the breast, to be opened by Dr. H. M. Moran (Sydney), followed by Dr. R. A. Gardner (Cairo), Dr. R. Kaye Scott (Melbourne), Dr. S. Verco (South Australia). Discussion on radiation treatment in carcinoma of the tongue, to be opened by Dr. R. A. Gardner (Cairo), followed by Dr. H. M. Moran (Sydney), Dr. L. J. Clendinnen (Melbourne).

Thursday, September 12.

10 a.m.—Discussion on radiological diagnosis in diseases of the lung, to be opened by Dr. J. G. Edwards (Sydney), followed by Dr. John O'Sullivan (Melbourne), Dr. B. L. W. Clarke (Brisbane). Discussion on some notes of the diagnosis of bone tumours, to be opened by Dr. H. R. Sear (Sydney), followed by Dr. Val. McDowell (Brisbane), Dr. Colin Macdonald (Melbourne), Dr. Howard Praagst (Melbourne).

Friday, September 13.

10 a.m.—Discussion on radiological education, to be opened by Major D. B. McGrigor (Frinton-on-Sea), followed by Dr. K. S. Cross (Melbourne), Dr. K. Hallam (Melbourne). Discussion on the radiological examination of the stomach and duodenum, to be opened by Dr. K. S. Cross, followed by Dr. John O'Sullivan (Melbourne), Dr. H. A. McCoy (Adelaide), Dr. de Moncheux (Dunedin).

SECTION OF DISEASES OF CHILDREN.

Office-Bearers.

President: Dr. Robert Hutchison, LL.D., M.D., F.R.C.P. (London).

Vice-Presidents: Dr. E. H. M. Stephen, M.B. (Sydney), Dr. H. Douglas Stephens, M.D., M.S., F.R.A.C.S. (Melbourne). Two vice-presidents to be appointed.

Honorary Secretaries: Dr. J. G. Whitaker, M.D., M.S., F.R.C.S. (55, Collins Street, Melbourne), Dr. Ian J. Wood, M.D., M.R.C.P. (12, Collins Street, Melbourne). English secretary to be appointed.

Programme.

Wednesday, September 11.

10 a.m.—Discussion on hare lip, to be opened by Dr. H. Douglas Stephens (Melbourne). Discussion on infant feeding, to be opened by Dr. H. Boyd Graham, followed by Dr. E. H. Williams (Dunedin, N.Z.), Dr. F. N. Le Messurier (Adelaide).

Friday, September 13.

10 a.m.—Discussion on pink disease, to be opened by Dr. A. Jeffreys Wood (Melbourne) and Dr. Ian J. Wood (Melbourne), followed by Dr. R. Hutchison (London), Dr. Edgar Stephen (Sydney), Dr. S. F. McDonald (Brisbane). Discussion on intussusception, to be opened by Dr. P. L. Hipsley (Sydney), followed by Dr. W. Vickers (Sydney), Dr. H. C. Colville (Melbourne).

SECTION OF NEUROLOGY AND PSYCHOLOGICAL MEDICINE.

Office-Bearers.

President: Professor Edwin Bramwell, M.D., F.R.C.P. Edin., F.R.C.P. (Edinburgh).

Vice-Presidents: Dr. A. W. Campbell, M.D. (Sydney), Dr. Bernard Hart, M.D., F.R.C.P. (London), Professor J. P. Lowson, M.D. (Brisbane). One vice-president to be appointed.

Honorary Secretaries: Dr. L. B. Cox, M.D., M.R.C.P. (37, Toorak Road, Malvern, S.E.5, Victoria), Dr. H. F. Maudsley, M.C., M.D. (8, Collins Street, Melbourne), Dr. J. K. Slater, M.B., F.R.C.P. (7 Walker Street, Edinburgh).

Programme.

Wednesday, September 11.

10 a.m.—Discussion on diagnosis, prognosis and treatment of brain tumours fifty years ago and now, to be opened by Professor Edwin Bramwell (Edinburgh), followed by Sir E. Farquhar Buzzard (Oxford), Sir James Purves Stewart (London), Professor Bouman (Amsterdam), Dr. H. M. Traquair (Edinburgh), Dr. A. W. Campbell (Sydney).

Friday, September 13.

10 a.m.—Discussion on psychoses of adolescence, to be opened by Professor W. S. Dawson (Sydney), followed by Dr. A. W. Campbell (Sydney), Dr. Evan Jones (Sydney), Dr. J. K. Adey (Melbourne), Dr. C. Farran-Ridge (Melbourne).

SECTION OF OPHTHALMOLOGY.

Office-Bearers.

President: Dr. A. J. Ballantyne, M.D., F.R.F.P.S. (Glasgow).

Vice-Presidents: Dr. J. Lockhart Gibson, M.D., F.R.A.C.S. (Brisbane), Dr. F. G. Antill Pockley, M.B., M.S. (Sydney), Dr. H. M. Traquair, M.D., F.R.C.S. (Edinburgh).

Honorary Secretaries: Dr. J. Ringland Anderson, M.C., M.B., B.Ch. (108, Collins Street, Melbourne, C.1), Dr. Max Yuille, M.B., F.R.C.S., D.O.M.S. (12, Collins Street, Melbourne), Dr. J. H. Doggart, M.D., F.R.C.S. (49, Wimpole Street, London, W.1).

Programme.

Thursday, September 12.

10 a.m.—Discussion on the ætiology, early diagnosis and medical aspects of glaucoma, to be opened by Dr. H. M. Traquair (Edinburgh), followed by Dr. A. J. Ballantyne (Glasgow), Dr. J. H. Doggart (London). Discussion on abnormal retinal correspondence and other hindrances to fusion training, to be opened by Dr. N. Bishop Harman (London) and Dr. T. a'B. Travers (Melbourne).

Friday, September 13.

10 a.m.—Discussion on cyclitis in female patients, to be opened by Dr. J. H. Doggart (London).

SECTION OF ORTHOPÆDICS.

Office-Bearers.

President: Professor E. W. Hey Groves, M.D., M.S., F.R.C.S. (Bristol).

Vice-Presidents: Dr. L. O. Betts, O.B.E., M.B., M.Ch. (Adelaide), Dr. S. T. Irwin, M.Ch., F.R.C.S. (Belfast), Dr. S. Alan S. Malkin, M.B., F.R.C.S. (54, The Ropewalk, Nottingham) (also acting as home secretary), Dr. J. Renfrew White, M.B., F.R.C.S. (Dunedin, N.Z.).

Honorary Secretaries: Dr. Thomas King, M.D., F.R.C.S. (2, Collins Street, Melbourne). English secretary to be appointed.

Programme.

Thursday, September 12.

10 a.m.—Discussion on fractures of the neck of the femur, to be opened by Professor E. W. Hey Groves (Bristol), followed by Dr. S. T. Irwin (Belfast). Discussion on

osteoarthritis of the knee and hip, to be opened by Dr. J. Forbes Mackenzie (Melbourne) and Dr. S. A. S. Malkin (Nottingham).

Friday, September 13.

10 a.m.—Discussion on fractures of the spine, to be opened by Dr. S. T. Irwin (Belfast), followed by Dr. F. C. Pybus (Newcastle-on-Tyne). Discussion on the rôle of physiotherapy in the treatment of injuries in general and orthopædic practice, to be opened by Dr. E. B. M. Vance (Sydney), followed by Dr. S. A. S. Malkin (Nottingham).

SECTION OF OTO-RHINO-LARYNGOLOGY.

Office-Bearers.

President: Dr. Francis Muecke, C.B.E., M.B., F.R.C.S. (London).

Vice-Presidents: Dr. J. Stoddart Barr, M.D., F.A.C.S. (Hobart), Dr. L. Graham Brown, M.C., M.D., F.R.C.S. (London), Dr. Douglas Guthrie, M.D., F.R.C.S. (Edinburgh) (also acting as home secretary), Dr. J. F. O'Malley, M.Ch., F.R.C.S. (London), Dr. W. N. Robertson, C.M.G., C.B.E., M.B., M.S., F.R.A.C.S (Brisbane).

Honorary Secretaries: Dr. G. A. D. McArthur, M.D., F.R.A.C.S. (85, Spring Street, Melbourne), Dr. J. H. Shaw, M.B., F.R.C.S., D.L.O. (55, Collins Street, Melbourne), Dr. Douglas Guthrie, M.D., F.R.C.S. (4, Rothesay Place, Edinburgh).

Programme.

Wednesday, September 11.

10 a.m.—Discussion on treatment of antral sinusitis, to be opened by Dr. J. F. O'Malley (London), followed by Dr. J. F. Woodburn (Sydney), Dr. Edgar Brown (Adelaide), Dr. E. A. Peters (London). Occasional papers: "Nasal Sinusitis in Children", by Dr. G. C. Scantlebury; "Sphenoidal Sinusitis and the Pituitary Gland", by Dr. C. M. Eadie.

Thursday, September 12.

10 a.m.—Discussion on the treatment of chronic suppurative *otitis media*, to be opened by Dr. L. Graham Brown (London), followed by Dr. Robert Godsall (Sydney), Dr. H. J. Gray (Perth, W.A.), Dr. Douglas Guthrie (Edinburgh). Occasional paper: "The Manifestations of Nasal Allergy: Their Diagnosis and Treatment", by Dr. H. M. Jay (Adelaide), followed by Dr. Ivan Maxwell (Melbourne).

SECTION OF PATHOLOGY AND BACTERIOLOGY.

Office-Bearers.

President: Professor A. Murray Drennan, M.D., F.R.C.P. (Edinburgh).

Vice-Presidents: Professor J. B. Cleland, M.D., Ch.M. (Adelaide), Dr. W. Keith Inglis, M.D., Ch.M. (Sydney). Two vice-presidents to be appointed.

Honorary Secretaries: Dr. C. H. Kellaway, M.C., M.D., M.S., F.R.C.P. (Melbourne Hospital, Melbourne), Professor H. A. Woodruff, M.R.C.S., L.R.C.P. (48, Fellowes Street, Kew, E.4, Victoria). English secretary to be appointed.

Programme.

Wednesday, September 11.

10 a.m.—Discussion on calcification, to be opened by Dr. F. Hansman (Sydney), followed by Professor P. MacCallum (Melbourne), Dr. Edgar King (Melbourne). Discussion on the pathology of osseous tissue, to be opened by Professor A. M. Drennan (Edinburgh), followed by Professor P. MacCallum (Melbourne), Dr. Keith Inglis (Sydney), Dr. Edgar King (Melbourne), Dr. R. Willis (Melbourne). Occasional papers: "Leuchæmic Infiltrations", by Professor J. B. Cleland (Adelaide); "Neural Components in Teratomata", by Dr. R. Willis (Melbourne).

Thursday, September 12.

10 a.m.—Discussion on anaerobes in disease, to be opened by Professor H. A. Woodruff (Melbourne), followed by Professor C. E. Hercus (Dunedin, N.Z.), Dr. W. J. Penfold (Melbourne), Professor H. A. Woodruff (Melbourne), Dr. G. H. Oxer (Mount Lawley, W.A.), Dr. C. W. Adey (Melbourne). Discussion on problems in virus disease, to be opened by Dr. F. M. Burnet (Melbourne), followed by Professor H. K. Ward (Sydney), Dr. Lionel Bull (Melbourne), Dr. E. V. Keogh (Melbourne). Occasional paper: "Determination of Hæmoglobin as Globin Picrate", by Dr. A. Bolliger (Sydney).

SECTION OF PHARMACOLOGY, THERAPEUTICS AND ANÆSTHESIA.

Office-Bearers.

President: Sir William Willcox, K.C.I.E., C.B., C.M.G., M.D., F.R.C.P. (London).

Vice-Presidents: Dr. L. A. Ivan Maxwell, M.D. (Melbourne), Dr. Z. Mennell, M.B. (London), Professor Sydney Smith, M.D., F.R.C.P.E., F.R.C.S.E. (Edinburgh), Dr. G. R. Troup, M.B., M.R.C.P. (Perth, W.A.).

Honorary Secretaries: Dr. Geoffrey Kaye, M.D. (14, Collins Street, Melbourne), Dr. B. L. Stanton, M.B., M.R.C.P. ("Rotha,", 95, Broadway, Camberwell, Victoria), Dr. E. Lewis Lilley, M.B., F.R.C.S. (Waterloo Gates, 86, New Walk, Leicester).

Programme.

Thursday, September 12.

10 a.m.—Discussion on the use and abuse of hypnotic drugs, to be opened by Sir William Willcox (London). Discussion on urinary antiseptics, to be opened by Dr. John Tait (Melbourne). Occasional paper: "A Vaso-Constrictor Principle in the skin of the Frog, *Hyla aurea*", by Professor W. A. Osborne (Melbourne).

Friday, September 13.

10 a.m.—Discussion on premedication and basal narcosis, to be opened by Dr. F. W. Green (Melbourne). Discussion on gas anæsthesia, to be opened by Dr. G. Kaye (Melbourne). Discussion on the position of spinal anæsthesia in Australia (opener not yet determined).

SECTION OF PUBLIC MEDICINE (TUBERCULOSIS, INDUSTRIAL AND TROPICAL HYGIENE) AND INCLUDING THE HISTORY OF THE DEVELOPMENT OF MEDICINE IN AUSTRALIA.

Office-Bearers.

President: Sir Henry Gauvain, M.D., M.Ch., F.R.C.S. (Alton).

Vice-Presidents: Sir R. W. Cilento, M.D. (Brisbane), Dr. C. E. Hercus, D.S.O., M.B., Ch.B. (Dunedin, N.Z.), Dr. G. Carmichael Low, M.D., F.R.C.P. (London). (One English vice-president to be appointed).

Honorary Secretaries: Dr. H. M. James, M.B., Ch.B. (22, Mayfield Avenue, Malvern, S.E.4, Victoria), Dr. F. R. Kerr, D.S.O., M.D., D.P.H. (27, Monomeith Avenue, Canterbury, E.7, Victoria), Professor R. H. Parry, M.D., M.R.C.P., D.P.H. (Bristol Health Offices, 40, Prince Street, Bristol).

Programme.

Wednesday, September 11.

10 a.m.—Discussion on the incidence of pleural effusion in artificial pneumothorax, with special reference to medical treatment, to be opened by Dr. D. B. Rosenthal (Melbourne). Discussion on the surgical treatment of purulent effusions in the chest, to be opened by Dr. M. P. Susman (Sydney).

Friday, September 13.

10 a.m.—(Combined meeting with the Section of Medical Sociology.) Discussion on racial pressure problems in Australia and neighbourhood, to be opened by Sir R. W. Cilento (Brisbane). Discussion on tropical medicine, to be opened by Dr. G. C. Low (London), followed by Dr. C. L. Park (Singapore). Discussion on Weil's disease, to be opened by Dr. T. J. Cotter (Queensland).

SECTION OF DERMATOLOGY.

Office-Bearers.

President: Dr. S. Watson Smith, M.D., F.R.C.P., M.R.C.P. (Bournemouth).

Vice-Presidents: Dr. L. P. Johnston, M.B., M.S. (Sydney), Dr. Herman F. Lawrence, M.R.C.P. (Melbourne). Two vice-presidents to be appointed.

Honorary Secretaries: Dr. R. R. Wettenhall, M.B., Ch.B. (85, Spring Street, Melbourne, C.1). English secretary to be appointed.

Programme.

Wednesday, September 11.

10 a.m.—Discussion on the incidence of skin diseases in Australia, to be opened by Dr. Herman Lawrence (Melbourne), followed by Dr. J. E. McGlashan (Perth, W.A.), Dr. W. C. T. Upton (Adelaide). Discussion on staphylococcal infections of the skin and their treatment, to be opened by Dr. J. Ivan Connor. Discussion on some aspects of mycological infections and their treatment, to be opened by Dr. J. C. Belisario (Sydney), followed by Dr. J. Witton Flynn (Sydney).

SECTION OF MEDICAL SOCIOLOGY.

Office-Bearers.

President: Dr. E. Kaye Le Fleming, M.D. (Wimbourne).
Vice-Presidents: Dr. D. G. Croll, C.B.E., M.B. (Brisbane), Reverend John Flynn, O.B.E. (Sydney), Professor J. A. Gunn, B.Sc., M.A., Ph.D. (Melbourne), James McRae, M.A., Esq. (Malvern, Victoria), Dr. Henry Robinson, M.D. (London).
Honorary Secretaries: Dr. George Simpson, M.B., M.R.C.P. (Heidelberg Road, Ivanhoe, N.21, Victoria), Dr. L. Dougal Callander, M.D. (Danum House, 6A, South Parade, Doncaster).

Programme.

Thursday, September 12.

10 a.m.—Discussion on Australian aerial medical services, to be opened by Dr. Allan Vickers (New South Wales), followed by Reverend John Flynn. Discussion on social aims of mental hygiene, to be opened by Professor Harvey Sutton (Sydney), followed by Professor J. A. Gunn (Melbourne), Dr. R. S. Ellery (Melbourne), Mrs. James McRae (Melbourne).

Friday, September 13.

10 a.m.—(Combined meeting with Section of Public Medicine *et cetera*.) Discussion on racial pressure problems in Australia and neighbourhood, to be opened by Sir R. W. Cilento (Brisbane).

PROVISIONAL TIME-TABLE.

Monday, September 9.

9 a.m. to 6 p.m.—Ladies' reception rooms open at the Oriental Hotel, Collins Street, Melbourne.
9 a.m. to 5 p.m.—Registration rooms open at the University of Melbourne. (Tickets for excursions for Monday, Tuesday and Wednesday; tickets for annual dinner; tickets for National Temperance League's annual dinner; tickets for Irish Medical Schools and Graduates' Association luncheon; tickets for medical missionary breakfast.)
2 p.m.—Official opening of trade exhibition.
2.30 p.m.—Short excursions by car *et cetera* in city and suburbs. Late afternoon parties and a number of small private dinner parties *et cetera* to visiting members.
8.30 p.m.—Reception by Victorian Medical Women's Society.

Tuesday, September 10.

9 a.m. to 5 p.m.—Registration rooms open. (All tickets available, except for excursions for Thursday, Friday and Saturday.) Ladies' reception room open.
10 a.m.—Ladies' excursions. Pathological museum open. Hobbies exhibition open.
11 a.m.—Adjourned annual general meeting in Town Hall, Melbourne. Installation of President of British Medical Association, 1935-1936. President's address.
2.30 p.m.—General excursions.
4.30 p.m.—Official religious service at Saint Paul's Cathedral. Late afternoon parties.
8.30 p.m.—Presidential reception.

Wednesday, September 11.

8.45 a.m.—Clinical address.
9 a.m. to 5 p.m.—Registration rooms open. (Excursion tickets for Thursday, Friday and Saturday available.) Ladies' reception room open. Trade exhibition open. Hobbies exhibition open.
10 a.m. to 1 p.m.—Scientific sections. Ladies' excursions.
1 p.m.—Irish Medical Schools and Graduates' Association luncheon.
2.30 p.m.—General excursions.
3 p.m.—Garden party at the University of Melbourne.
5.30 p.m.—Late afternoon parties.
9.30 p.m.—Reception at Government House by Their Excellencies Lord and Lady Huntingfield.

Thursday, September 12.

8.30 a.m.—National Temperance League breakfast. Host: Dr. J. F. Mackeddie. Mr. W. McAdam Eccles will deliver the address.
8.45 a.m.—Clinical address.
9 a.m. to 5 p.m.—Registration rooms open. Ladies' reception rooms open. Trade exhibition open. Pathological museum open. Hobbies exhibition open.
10 a.m. to 1 p.m.—Scientific sections. Ladies' excursions.
2.30 p.m.—General excursions.
5.30 p.m.—Late afternoon parties.
7.15 p.m.—Annual dinner (limited to about 800 members of the Association).
9 p.m.—Ball given by members of the Victorian Branch at the Town Hall, Melbourne.

Friday, September 13.

8.30 a.m.—Medical missionary breakfast.
8.45 a.m.—Clinical address.
9 a.m. to 5 p.m.—Registration rooms open. Trade exhibition open. Pathological museum open. Hobbies exhibition open.
9.30 a.m.—Notts Ladies' Challenge.
10 a.m.—Ladies' excursions.
10 a.m. to 1 p.m.—Scientific sections.
2.30 p.m.—General excursions.
5.30 p.m.—Late afternoon parties.
8 p.m.—Popular lecture.
8.30 p.m.—Army, Navy and Air Force Medical Services' Ball (by invitation). Members of the Royal Australian Naval Medical Service and Reserve, Australian Army Medical Corps and Reserve, Royal Australian Air Force Medical Service and Reserve will be the hosts.

HOTEL AND BOARDING HOUSE ACCOMMODATION.

Below will be found a list of hotels and boarding houses in Melbourne recommended for the accommodation of visitors to the annual meeting. Application should be made direct to the hotels and boarding houses, mentioning the British Medical Association.

Name.	Total Accommodation.	Charges.			
		Bed and Breakfast.	Inclusive Tariff.	Luncheon.	Dinner.
MELBOURNE.					
Menzies' Hotel	220	16/- day	27/- day	5/-	8/6
Hotel Alexander ..	350	15/- day	£5/5/- wk.	3/6	6/6
Hotel Windsor	300	12/6 day 16/6 day	21/- day 25/- day	4/-	6/6
Oriental Hotel	120	10/6 day	20/- day	4/-	7/6
Scott's Hotel	180	11/6 day	21/6 day	*à la carte*	
Federal Hotel	500	7/- day 12/- day	15/- day 21/- day	3/6	5/-
Victoria Palace (unlicensed)	850	5/- day 10/- day	8/- day 17/6 day	*à la carte*	
New Treasury Hotel	45	8/6 day	15/- day		
ST. KILDA.					
George Hotel	125	9/- day 13/6 day	£5/5/- wk.	4/6	7/-
Esplanade Hotel ..	120	9/- day	20/- day	3/-	4/-
Chevron (unlicensed)	200	10/6 day	15/- day	3/-	4/-

These charges may be subject to slight alteration. All the hotels of Melbourne are approximately three-quarters of a mile to one mile from the University. There are a number of small unlicensed guest houses situated in the environs of Melbourne, details of which are as yet unavailable.

THE GEORGE MACDONALD TESTIMONIAL FUND.

THE undermentioned subscriptions have been received for the George Macdonald Testimonial Fund:

£5 5s.: Dr. S. S. Gardiner, Dr. B. T. Edye. (Dr. Edye's contribution was incorrectly stated in last week's journal as being £2 2s.)
£2 10s.: Fifth Year Medical Students, University of Sydney.
£2 2s.: Dr. D. Miller, Dr. C. S. Graham, Dr. B. W. B. Riley, Dr. G. A. M. Heydon, Dr. A. C. Manning, Dr. T. Ewing, Dr. N. Paul.
£1 10s.: Mr. Louis Schaeffer.
£1 9s. 6d.: Fourth Year Medical Students, University of Sydney.
£1 9s.: Sixth Year Medical Students, University of Sydney.
£1 7s.: Department of Engineering, University of Sydney.
£1 2s.: Department of Physics, University of Sydney.
£1 1s.: Professor H. Priestley, Dr. N. W. G. Macintosh, Dr. W. R. Wilson, Dr. A. E. Burrell, Dr. J. G. Stephens, Dr. A. S. Boyd, Dr. D. J. Glissan, Professor J. C. Windeyer, Dr. H. Solomon, Dr. Lorna D. Beveridge, Dr. Kathleen Winning, Dr. A. C. Thomas, Dr. P. Walton Smith, Mr. J. Shewan, Mr. R. S. Freeman, Dr. K. S. Wallace, Dr. W. F. Simmons, Dr. J. O'Donnell, Dr. M. B. McIlrath, Dr. W. L. Magill.
£1: Dr. N. E. Goldsworthy, Dr. F. N. Chenhall, Dr. H. G. McQuiggin.
10s. 6d.: Dr. C. W. Dun, Dr. W. G. English, Sir J. B. Peden, Dr. L. Bamber, Dr. A. W. J. Bulteau, Dr. A. H. Moseley, Dr. G. Cummins, Dr. E. S. Stuckey, Mr. G. R. King.
10s.: Dr. H. V. Wilson, Mr. H. Clarke, Mr. E. Collins, Department of Geology, University of Sydney.

Diary for the Month.

JUNE 4.—Tasmanian Branch, B.M.A.: Council.
JUNE 5.—Western Australian Branch, B.M.A.: Council.
JUNE 5.—Victorian Branch, B.M.A.: Branch.
JUNE 6.—South Australian Branch, B.M.A.: Council.
JUNE 11.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JUNE 11.—Tasmanian Branch, B.M.A.: Branch.
JUNE 14.—Queensland Branch, B.M.A.: Council.
JUNE 15.—Victorian Branch, B.M.A.: Branch.
JUNE 18.—Tasmanian Branch, B.M.A.: Council.
JUNE 18.—New South Wales Branch, B.M.A.: Ethics Committee.
JUNE 19.—Victorian Branch, B.M.A.: Clinical Meeting.
JUNE 19.—Western Australian Branch, B.M.A.: Branch.
JUNE 20.—New South Wales Branch, B.M.A.: Clinical Meeting.
JUNE 25.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JUNE 27.—South Australian Branch, B.M.A.: Branch.
JUNE 27.—New South Wales Branch, B.M.A.: Branch.
JUNE 28.—Queensland Branch, B.M.A.: Council.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xviii, xix. and xxi.

CHILDREN'S HOSPITAL (INCORPORATED), PERTH, WESTERN AUSTRALIA: Junior Resident Medical Officer.
CENTRAL EYRE PENINSULA DISTRICT HOSPITAL (INCORPORATED), WUDINNA, SOUTH AUSTRALIA: Medical Officer.
COMMONWEALTH DEPARTMENT OF HEALTH, CANBERRA, FEDERAL CAPITAL TERRITORY: Medical Assistant.
LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officers.
LEWISHAM HOSPITAL, LEWISHAM, NEW SOUTH WALES: Honorary Radiologist.
PRINCE HENRY HOSPITAL, RANDWICK, NEW SOUTH WALES: Honorary Consulting Thoracic Surgeon.
RENWICK HOSPITAL FOR INFANTS, SUMMER HILL, NEW SOUTH WALES: Resident Medical Officer.
ROYAL AUSTRALIAN AIR FORCE: Medical Officer.
THE WOMEN'S HOSPITAL, CROWN STREET, SYDNEY, NEW SOUTH WALES: Junior Resident Medical Officers.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associate Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointment and those desiring to accept appointments to any COUNTRY HOSPITAL, are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	Officer of Health, District Council of Elliston. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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